October 1991
Volume 36, Number 10
ISSN 0098-1142-RECACP

RESPIRATORY CARE

A MONTHLY SCIENCE JOURNAL
36TH YEAR—ESTABLISHED 1956

- Professional Literacy
- Determination of Ventilatory Reserve in Mechanically Ventilated Patients
- Effect of PEEP-Valve Function on a Home-Care Ventilator
- Cost Impact of MDI vs SVN: Cleveland Clinic Experience
- Comparison of Ventilation Delivered with Pocket Mask vs Demand Valve
- Flow-By vs T-Piece Ventilation prior to Extubation in Postsurgical Patients
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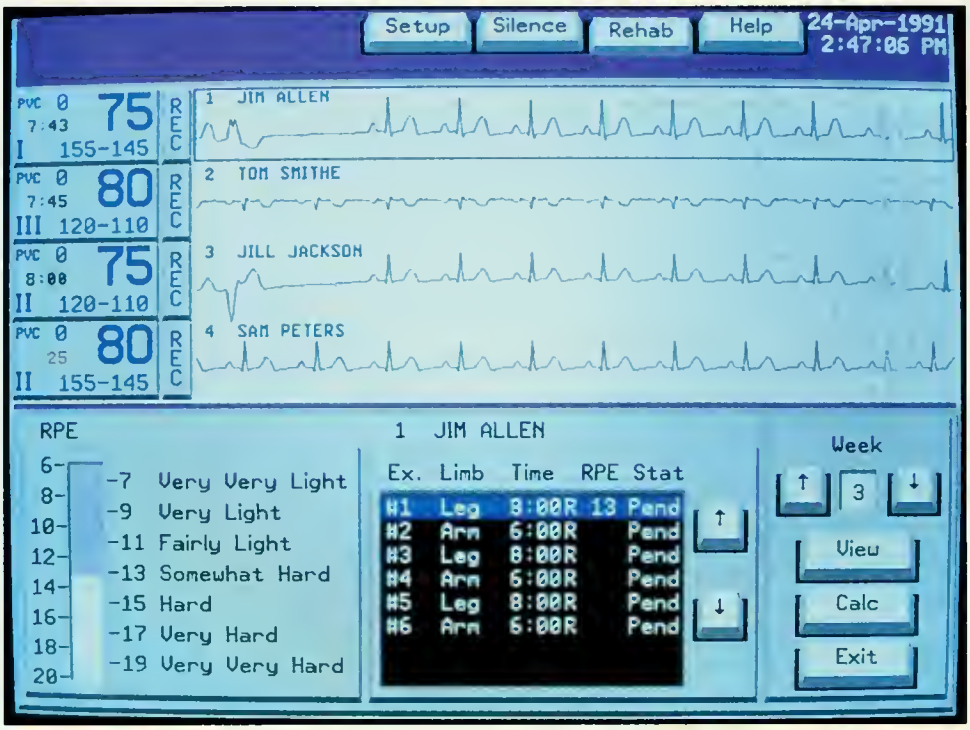

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Association of *Chlamydia pneumoniae* (Strain TWAR) Infection with Wheezing, Asthmatic Bronchitis, and Adult-Onset Asthma—DL Hahn, RW Dodge, R Golubjatnikov. JAMA 1991;266:225.

OBJECTIVE: to study the clinical characteristics of respiratory tract illness caused by *Chlamydia pneumoniae* (*C pneumoniae*). DESIGN: prospective clinical, bacteriologic, and serologic study. Secondarily, a matched comparison of patients with and without evidence of *C pneumoniae* infection (serologic titers ≥ 1:64 and < 1:16, respectively). SETTING: four primary care (family practice) clinics in Madison, Wisconsin, and nearby towns. PATIENTS: the study included 365 white males and females (mean age 34.2 years). MAIN OUTCOME MEASURES: association of acute *C pneumoniae* infection with signs and symptoms of respiratory illness and the relationship of *C pneumoniae* antibody titer with wheezing at the time of enrollment in the study, and with the diagnosis of asthmatic bronchitis. RESULTS: nine (47%) of 19 patients with acute *C pneumoniae* infection had bronchospasm during respiratory illness, and there was a strong quantitative association of *C pneumoniae* titer with wheezing at the time of enrollment in the study ($p = 0.01$). In the matched study, *C pneumoniae* antibody was significantly associated with asthmatic bronchitis after, but not before, respiratory illness (odds ratio, 7.2; 95% confidence interval, 2.2 to 23.4). Four infected patients had newly diagnosed asthma after illness, and four others had exacerbation of previously diagnosed asthma. There was no serologic evidence of coexisting *Mycoplasma pneumoniae*, *Chlamydia trachomatis*, or respiratory viral infection in 96% of patients with asthmatic bronchitis and asthma. CONCLUSIONS: some *C pneumoniae* antibody titers, although not diagnostic of chlamydial infection by present criteria, probably represent acute reinfection or ongoing chronic infection. Repeated or prolonged exposure to *C pneumoniae* may have a causal association with wheezing, asthmatic bronchitis, and asthma.

Shortcomings of Using Two Jet Nebulizers in Tandem with an Aerosol Face Mask for Optimal Oxygen Therapy—GN Foust, WA Potter, MD Wilons, EB Golden. Chest 1991;99:1346.

Herein, a laboratory model that allows measurement of simulated distal airway oxygen percentage at different breathing patterns is described to illustrate the shortcomings of conventional O_2 devices and, in particular, the aerosol face mask with two jet nebulizers (AFM-DF) in tandem. A table showing the degree of dilution that occurred during simulation of various breathing patterns while using AFM-DF is also presented. Data revealed that when 60% was desired, 13 of 27 measurements were less than 55%. The worst-case scenario for 60% desired was 48% measured. When 80% was desired, less than 70% was delivered in 24 of the 27 breathing patterns simulated. Less than 60% was measured on 12 occasions, with 51% being the lowest measurement. When 100% O_2 was desired, less than 80% was measured in 25 of 27 breathing patterns. Less than 60% was measured in 10 of those. Fifty percent was the lowest analyzed value for the 100% setting. The inadequacy of AFM-DF is described in three case studies. A high-flow nonrebreathing face mask (HFM) to address the subset of patients is also discussed. A peak inspiratory flow prediction chart is also documented and may be useful in setting optimal flows when using high-flow systems. The patients in whom intubation and mechanical ventilation (or use of continuous mask CPAP) are indicated can be more clearly identified with a trial of high-flow O_2 therapy (with a system that assures adequate flow to meet the patient's peak inspiratory flow demands). In the remainder of patients, those higher-risk modalities will be precluded.

Changes in Mucociliary Clearance during Acute Exacerbations of Asthma—MS Messina, TG O'Riordan, GC Smaldone. Am Rev Respir Dis 1991;143:993.

Previous studies have suggested that mucociliary clearance (MC) is impaired in asthmatic subjects. If so, impaired clearance may be an important factor in acute exacerbation. We proposed that if MC plays a significant role in acute illness, MC should be impaired during the exacerbation but improve after recovery. To test this hypothesis, five asthmatic patients with attacks requiring hospitalization underwent measurement of MC using radiolabeled aerosol and a gamma camera. They were studied on the second or third day after admission with repeat measurements after discharge. Spirometry was performed before all studies. After an equilibrium xenon scan (^{133}Xe), which defined lung borders and measured regional volume, radiolabeled saline particles containing technetium-labeled (^{99m}Tc) sulfur colloid were deposited and used to label airway mucus. Deposition patterns were matched by regulating particle distribution and breathing pattern. MC was then measured as percentage retention of radioactivity at 10-min intervals for 2 hours. When hospitalized, 96.0 ±

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2.06% (SEM) of the initial radioactivity was retained in the lung after 2 hours, indicating little clearance of mucus from the lung. In fact, no significant changes were detected when activity at 120 min was compared with measurements at 10 min (99.2 ± 0.22%, NS). After discharge, clearance was markedly enhanced. That is, retention of lung activity was significantly lower at all time intervals from 10 min onward, with only 70.9 ± 3.86% retained at 120 min ($p = 0.008$). During an asthmatic attack warranting hospital admission, MC is significantly impaired, with marked improvement following recovery. Although a causal relationship remains to be established between mucociliary impairment and the clinical features of an acute exacerbation of asthma, the association demonstrated in this paper suggests that MC assessment may yet provide a further objective means of assessing the severity of a patient's asthma and the response to therapy.

The Effect of Maximal Doses of Formoterol and Salbutamol from a Metered Dose Inhaler on Pulse Rates, ECG, and Serum Potassium Concentrations—FPV Maesen, R Costongs, JJ Smeets, PJ Brombacher, PGMA Zweers. Chest 1991; 99:1367.

In a randomized, double-blind, crossover cumulative study, the individual maximal bronchodilator dosages for formoterol (F) and salbutamol (S) were assessed for their respective influence on ECG, pulse rate, and serum potassium levels in 13 patients with stable and reversible asthma. The following dosages were administered with an interval of 1 hour: 12-24-48-(48)-(48) µg for F and 100-200-400-400-(400)-(400) µg for S. The study day was discontinued if pulse rate was above 140 beats · min^{-1}, a flattening of T wave on the ECG was recorded, or a maximal bronchodilation in FEV_1 was observed (above 110% of the predicted value or an increase in FEV_1 in the last two measurements below 5%). The maximal individual dose of F administered was 84 µg in six patients, 132 µg in three patients, 180 µg in three patients, and 228 µg in one patient. For S, the maximal individual dose was 400 µg in three patients, 2,200 µg in eight patients, 3,000 µg in one patient, and 3,800 µg in one patient. The mean maximal increase in FEV_1 was 36.0% after F and 35.1% after S. Pulse rate increased from 73 to 83 beats · min^{-1} after F and from 75 to 84 beats · min^{-1} after S (both statistically significant). No pulse rate above 140 beats · min^{-1} was observed. In the high-therapeutic range (up to 36 µg of F and 6,090 µg of S), no changes in potassium level were observed. In still higher dosages, mean potassium level decreased from 4.16 to 3.78 mmol · L^{-1} after F and from 4.02 to 3.88 mmol · L^{-1} after S (not clinically relevant). The lowest individual potassium level recorded was 3.1 mmol · L^{-1}. No clinically important changes in ECG were observed. In conclusion, very high doses of F and S administered from a metered dose inhaler proved to be safe for patients.

Effect of Dexamethasone on Pulmonary Inflammation and Pulmonary Function of Ventilator-Dependent Infants with Bronchopulmonary Dysplasia—MC Yoder Jr, R Chua, R Tepper. Am Rev Respir Dis 1991;143:1044.

Seventeen ventilator-dependent premature infants with bronchopulmonary dysplasia (BPD) were enrolled in a double-blind, placebo-controlled study to determine the effect of 3 days of intravenously administered dexamethasone (0.5 mg/kg/day) on pulmonary function, pul-

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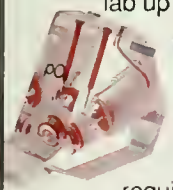

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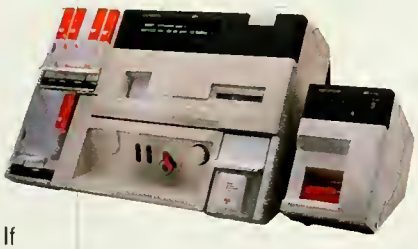

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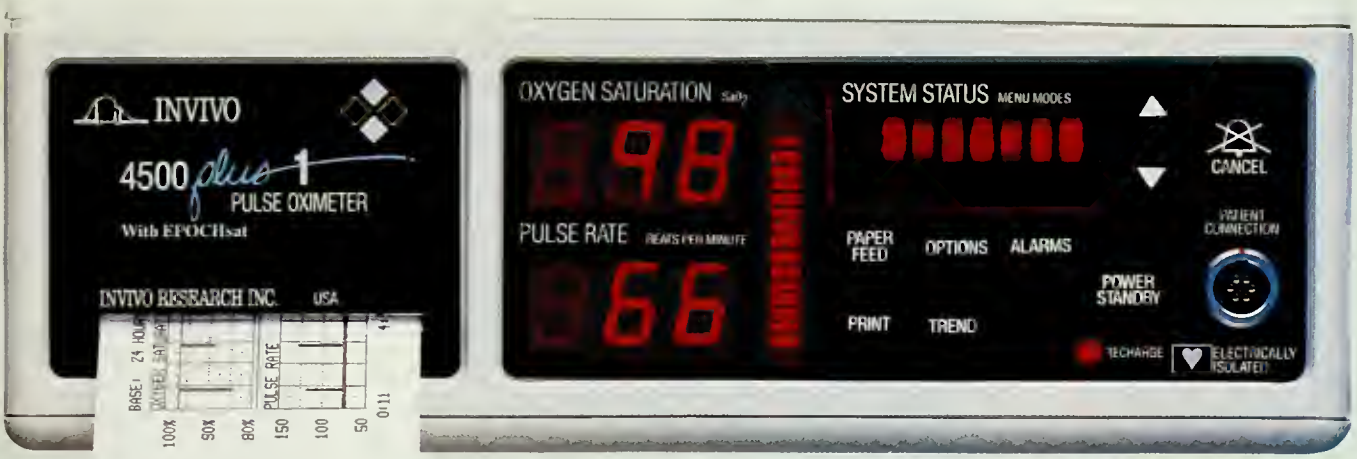
monary inflammation, and the requirement for respiratory support (F_{IO_2}, ventilator peak pressure [PP], and respiratory rate [RR]). Assessment of pulmonary function included measurement of FVC, flow at 25% vital capacity ($\dot{V}_{25}$), and static compliance of the respiratory system (C_{rs}), whereas pulmonary inflammation was assessed by the neutrophil count, ratio of elastase/2 × alpha$_1$-antitrypsin, and the concentrations of albumin and fibronectin in the tracheobronchial lavage (TBL) fluid. After 3 days of placebo treatment, there were no significant changes in any of the measured parameters. In contrast, the dexamethasone-treated group demonstrated a significant decrease in respiratory support (F_{IO_2}: 50 versus 36%; PP: 21 versus 16 cm H_2O; RR: 22 versus 14 breaths/min) and improved pulmonary function (C_{rs}: 0.63 versus 0.85 mL/cm H_2O/kg; $\dot{V}_{25}$: 23 versus 68 mL/s/kg). In addition, pulmonary inflammation was suppressed in the dexamethasone-treated group (neutrophils: 23 versus 11 × 10^3/mg albumin; elastase/2 × alpha$_1$-antitryp-sin: 0.24 versus 0.10; albumin: 7.1 versus 3.5 mg/dL; fibronectin: 33 versus 17 µg/mg albumin). We conclude that short-term treatment with dexamethasone improves pulmonary function and suppresses pulmonary inflammation as well as decreasing the respiratory support required by ventilator-dependent premature infants with BPD.

Smoking and Mortality among Older Men and Women in Three Communities—AZ LaCroix, J Lang, P Scherr, RB Wallace, J Cornoni-Huntley, L Berkman, et al. N Engl J Med 1991;324:1619.

BACKGROUND: Although cigarette smoking is the leading avoidable cause of premature death in middle age, some have claimed that no association is present among older persons. METHODS: We prospectively examined the relation of cigarette-smoking habits with mortality from all causes, cardiovascular causes, and cancer among 7,178 persons 65 years of age or older without a history of myocardial infarction, stroke, or cancer who lived in one of three communities: East Boston, Massachusetts; Iowa and Washington counties, Iowa; and New Haven, Connecticut. At the time of the initial interview, prevalence rates of smoking in the three communities ranged from 5.2 to 17.8% among women and from 14.2 to 25.8% among men. During 5 years of follow-up, there were 1,442 deaths, 729 due to cardiovascular disease, and 316 due to cancer. RESULTS: In both sexes, rates of total mortality among current smokers were twice what they were among participants who had never smoked. Relative risks, as adjusted for age and community, were 2.1 among the men (95% confidence interval, 1.7 to 2.7) and 1.8 among the women (95% confidence interval, 1.4 to 2.4). Current smokers had higher rates of cardiovascular mortality than those who had never smoked (as adjusted for age and community, the relative risk was 2.0 [95% confidence interval, 1.4 to 2.9] among the men and 1.6 [95% confidence interval, 1.1 to 2.3] among the women), as well as increased rates of cancer mortality (relative risk, 2.4 [95% confidence interval, 1.4 to 4.1] among the men and 2.4 [95% confidence interval, 1.4 to 3.9] among the women). In both sexes, former smokers had rates of cardiovascular mortality similar to those of the participants who had never smoked, regardless of age at cessation, whereas the rates for all cancers, as well as smoking-related cancers, remained elevated among men who had once smoked. CONCLUSIONS: Our prospective findings indicate that the mortality hazards of smoking extend well into later life, and suggest that cessation will continue to improve life expectancy in older people.

Relationship of Airway Hyperresponsiveness to Respiratory Symptoms and Diurnal Peak Flow Variation in Patients with Obstructive Lung Disease—PLP Brand, DS Postma, HAM Kerstjens, GH Koëter, and the Dutch CNSLD Study Group. Am Rev Respir Dis 1991;143:916.

This study reports on the relationship of airway hyperresponsiveness (AH) with respiratory symptoms and diurnal peak expiratory flow (PEF) variation in 221 hyperresponsive patients with moderately severe airways obstruction. The disease was in a stable phase in all patients. Closely adhering to the American Thoracic Society criteria, patients were divided into three syndrome diagnoses based on a standardized history: asthma (n = 81), asthmatic bronchitis (AB, n = 69), and chronic obstructive pulmonary disease (COPD, n = 44); 27 subjects could not be placed in any group. Mean (± SEM) $\log_2$ PC_{20} histamine values were significantly lower in the asthmatic group (–2.77 ± 0.20 mg/mL) than in the COPD (–0.89 ± 0.29 mg/mL) and AB groups (–1.37 ± 0.25 mg/mL; one-way ANOVA, $p < 0.001$). However, considerable overlap of individual responses existed. Differences between the groups could not be attributed to differences in prechallenge FEV_1 levels. For every level of FEV_1, asthmatic subjects were more hyperresponsive than patients with COPD. The dependence of PC_{20} on prechallenge FEV_1 was comparable in all groups. There was a significant correlation between the degree of AH and diurnal PEF variation ($p = -0.401$, $p < 0.001$), which was stronger in asthma ($p = -0.409$) than in COPD ($p = -0.325$). Despite



SaO2 DATA SUMMARY

99 % OF TIME SaO2 90 - 100%	
0 % OF TIME SaO2 85 - 89%	
0 % OF TIME SaO2 80 - 84%	
0 % OF TIME SaO2 70 - 79%	
0 % OF TIME SaO2 < 70%	

EPOCHSAT STATISTICS

SAT OF HIGHEST OCCURANCE (NORMAL) = 97 %
DROP: 3% DURATION: 1 MIN. 0 SEC. BASE: 24 HR
TOTAL EPOCHSAT COUNT - 5
AVERAGE MINIMUM SaO2 DURING ALL EPOCHSAT EVENTS - 89
OF EPOCHSAT EVENTS LESS THAN 90% - 2
AVERAGE DURATION OF EPOCHSAT EVENTS - 2 MIN. 0 SEC.
AVERAGE RECOVERY OF EPOCHSAT EVENTS - 1 MIN 0 SEC.

EPOCHSAT EVENT #	LOG TIME	LOWEST SaO2	DURATION(T)	RECOVERY(T)	PR
1	01:01	85%	2 MIN. 0 SEC	1 MIN. 0 SEC	120
2	03:01	89%	2 MIN. 40 SEC	1 MIN. 40 SEC	130
3	04:31	91%	2 MIN. 40 SEC	1 MIN. 40 SEC	82
4	11:01	91%	2 MIN. 0 SEC	1 MIN. 0 SEC	82
5	17:01	90%	2 MIN 0 SEC	1 MIN. 0 SEC	100

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Introducing the desaturation monitor.

The Invivo 4500 Plus Pulse Oximeter Series with EPOCHsat changes the expectations for pulse oximetry in Respiratory Care. Now, assessing the adequacy of oxygenation is automatic — with the EPOCHsat continuous record and analysis of patient desaturation.

More than $Sp0_2$ storage and strip-chart printouts. More than intermittent human recordings and evaluations of continuous $Sp0_2$. EPOCHsat automatically monitors for desaturation events and analyzes key oxygenation conditions.

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Invivo. *The oximeter of choice with Respiratory Care Professionals.*

Nothing says more about the quality of a company — its products, services, and people — than the longevity of its customer relationships. In our 13 years in the cardiopulmonary market, Medical Graphics Corporation has established an enviable record of customer loyalty — ranging from the small clinic and physician's office, to large research hospitals, and even to NASA, who will be relying on MedGraphics quality during many of its Space Shuttle missions.

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MedGraphics

Over the years, we have worked closely with our customers to define the standards of quality. First and foremost, our systems answer the need for accuracy. Reliability, expandability, ease of use, and cost-effectiveness are also hallmarks of the MedGraphics name. These standards have guided the development of the MedGraphics Pulmonary Function Testing System 1070 and the Body Plethysmography System 1085 (shown here) which have come to be regarded worldwide as simply the best. Now, we are pleased to announce that both of these systems are available with our new generation of easier-than-ever-to-use software — BREEZE™. Also new — our Body Plethysmography System 1085D features complete lung function capabilities including diffusing capacity measurement.

The new MedGraphics BREEZE diagnostic software, with its colorful icons, makes testing a breeze!

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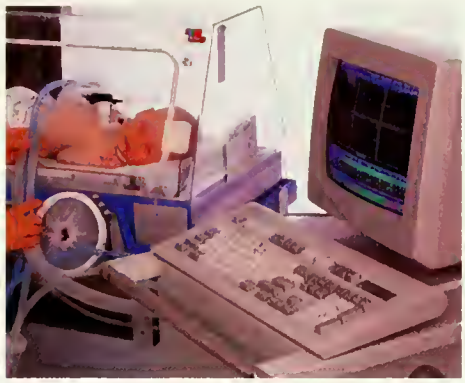

In truly critical situations, physicians rely on the MedGraphics CCM Critical Care Management System for vital metabolic measurements that can help avoid ventilatory complications arising from inappropriate nutrition. In the ICU, the MedGraphics CCM assesses nutrition and the mechanics of breathing to dramatically decrease ventilator dependence and significantly improve patient outcome. *Even neonates weighing as little as 500 grams can be accurately evaluated for ventilatory, metabolic and cardiac function, including cardiac output, with the MedGraphics Pediatric CARE™ System (shown here). Optimal feeding and ventilator management, made possible by MedGraphics' exclusive breath-by-breath gas exchange technology, can play an important role in increasing an infant's chances for survival and improving its long-term quality of life.*

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this obvious association, a wide range of diurnal PEF variation values existed for every level of PC_{20}, indicating that PEF variability and AH are not interchangeable. The relationships between symptoms and both AH levels and PEF variation were weak. No significant differences were found between syndrome diagnosis groups with respect to diurnal PEF variation. We hypothesize that symptoms, diurnal PEF variation, and AH may all provide different information on the actual disease state.

The Relationship between the Arterial to End-Tidal P_{CO_2} Difference and Hemoglobin Saturation in Patients with Congenital Heart Disease—R Fletcher. Anesthesiology 1991;75:210.

In right-to-left (RL) intracardiac shunting, the venous blood that is added to the oxygenated blood in the left heart is both poor in oxygen and rich in carbon dioxide. Thus, any given degree of arterial desaturation is associated with an obligatory arterial to end-tidal carbon dioxide tension difference ($P_{aCO_2} - P_{etCO_2}$). This paper presents a theoretical analysis of the relationship between $P_{aCO_2} - P_{etCO_2}$ and arterial hemoglobin saturation (S_{aO_2}) in cyanotic heart disease. Using the shunt equation as a starting point, a curvilinear, negative correlation between $P_{aCO_2} - P_{etCO_2}$ and S_{aO_2} can be demonstrated. The slope of the regression of $P_{aCO_2} - P_{etCO_2}$ against S_{aO_2} is shown to be positively correlated to Hb concentration, P_{aCO_2}, and the respiratory quotient R. The slope of the regression is also slightly increased at relatively high S_{aO_2}s and at high inspired oxygen fractions, although these latter factors are of lesser significance. However, in addition to the above primary effects of RL shunting, secondary effects may occur if pulmonary perfusion is reduced sufficiently to cause "alveolar hypoperfusion," which also creates an alveolar dead space. Primary and secondary effects are additive. This theoretical analysis is illustrated with a study of 27 children with congenital heart disease. Their lungs were ventilated with a Servo 900C ventilator, and carbon dioxide single-breath tests were obtained on-line with the use of a computerized system based on the Siemens-Elema carbon dioxide analyzer 930. Blood was sampled for P_{aCO_2} measurement and arterial Hb saturation was measured by pulse oximetry (S_{pO_2}). The relationship between $P_{aCO_2} - P_{etCO_2}$ and S_{pO_2} was found to agree with that predicted by theory, confirming that in cyanotic heart disease, $P_{aCO_2} - P_{etCO_2}$ increases by 0.2-0.4 kPa [2-3 torr] for every 10% reduction in S_{pO_2}. Awareness of this relationship is necessary when attempting to estimate P_{aCO_2} from P_{etCO_2} during anesthesia in cyanotic children.

Effect of Canister Temperature on Performance of Metered-Dose Inhalers—AF Wilson, DS Mukai, JJ Ahdout. Am Rev Respir Dis 1991; 143:1034.

We studied the effects of ambient temperatures between 0°C and 42°C in an anatomically realistic model of the upper airway ventilated with warm, moist air. Metered dose inhalers of metaproterenol maintained at the experimental temperatures were activated in the mouth of the model at the onset of inspiration. We measured the amount of drug released and that which was able to penetrate the model at each temperature. At low canister temperatures considerably more drug was released than at higher temperatures. However, at higher temperatures much more drug was able to traverse the upper airway, raising lower airway deposition from 17.7% at 4°C to 32.2% at 37°C. Aerosol size became progressively smaller as temperature was raised: mass median diameter (MMD) was 3.65 μm at 0°C and 2.5 μm at 37°C. These findings are most likely due to greater canister vapor pressures at higher temperatures. We conclude that patients should probably be instructed to prewarm metered-dose inhaler canisters prior to use when canister temperatures are low.

Effect of Low Concentrations of Ozone on Inhaled Allergen Responses in Asthmatic Subjects—NA Molfino, SC Wright, I Katz, S Tarlo, F Silverman, PA McClean, et al. Lancet 1991;338:199.

The relation between inhalation of ambient concentrations of ozone and airway reactivity to inhaled allergens may be important in asthma, since both agents can produce inflammatory changes in the airways. Seven asthmatic patients (mean age 40 [SD 13] years), with seasonal symptoms of asthma and positive skin tests for ragweed or grass, took part in a study to investigate whether exposure to low concentrations of ozone potentiates the airway allergic response. The patients were studied during 4 separate weeks in the winter. In each week there were 3 study days: on Days 1 and 3 methacholine challenges were carried out; and on Day 2 the subject received one of four combined challenges in a single-blind design—air breathing followed by inhalation of allergen diluent (placebo); ozone followed by inhalation of allergen diluent; air followed by allergen; or ozone followed by allergen. The ozone concentration was 0.12 ppm during 1 hour of tidal breathing at rest, and allergens were inhaled until the forced expiratory volume in 1 second (FEV_1) had fallen by 15% (PC_{15}). There were no

More hope for infants with RDS*
More proof of effectiveness
More experience
Baby's Breath
Gypsophila paniculata
™
*Respiratory distress syndrome

The proven efficacy and impressive safety profile of the first protein-free, synthetic lung surfactant

Exosurf NEONATAL

(Colfosceril Palmitate, USP, Hexadol, Tyloxapol) for intratracheal suspension

[illegible] clinical trials, with EXOSURF Neonatal proven [illegible] reduction in the incidence and mortality of infants with RDS. Successful use under a year-long [illegible] confirmed its efficacy and impressive safety profile. Since there is [illegible] of benefit over [illegible], widespread use is expected across the [illegible] has been established to [illegible] in the treatment of RDS. The weight of clinical [illegible] in favor of EXOSURF Neonatal.

Dramatic reductions in neonatal morbidity and mortality reported in clinical trials

[illegible] shown in clinical outcome after EXOSURF Neonatal has been significant in [illegible] of life in decreasing RDS as well as those with established RDS. Prompt [illegible] as rescue treatment with EXOSURF Neonatal has dramatically reduced [illegible] and mortality in infants weighing greater than 700 grams.

	Prophylactic treatment		Rescue treatment	
Age	Single Dose 700–1100 grams (N=[illegible])	Three Doses 700–1100 grams (N=[illegible])	Two Dose 700–1250 grams (N=[illegible])	Two Dose 1251 grams and above (N=[illegible])
[illegible]	—	—	[illegible]%	[illegible]%
[illegible]	[illegible]%	[illegible]%	[illegible]%	[illegible]%
[illegible]	[illegible]%	[illegible]%	[illegible]%	[illegible]%

[illegible] prophylactic dose of EXOSURF Neonatal reduced 1-year mortality by [illegible]% [illegible] clinical prophylactic doses provided an additional [illegible]% reduction in 1-year [illegible]

	Prophylactic treatment		Rescue treatment	
	Single Dose 700–1100 grams (N=[illegible])	Two Dose 700–1250 grams (N=[illegible])	Two Dose 1251 grams and above (N=[illegible])	
Reduction in death from RDS	[illegible]%	[illegible]%	[illegible]%	

[illegible] (1) [illegible] (2) [illegible] (3) [illegible] (4) [illegible] N=Number of infants enrolled in clinical

SIGNIFICANT REDUCTION IN CARDIOPULMONARY DESTRUCTION TO DAY 28 FOLLOWING TWO-DOSE RESCUE TREATMENT[4,5]

(Percent reductions with EXOSURF)

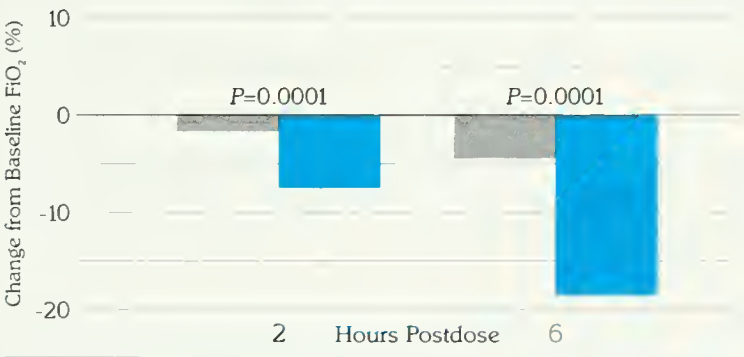
Birth weight	Reduction in death at 28 days or survival with broncho-pulmonary dysplasia (BPD)
700-1350[4] (N=419)	34%*
1250 and above[5] (N=1232)	43%*

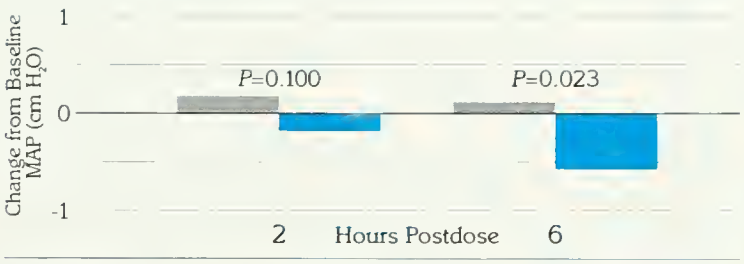
*P=0.002. N=Number of infants enrolled in the clinical trials.

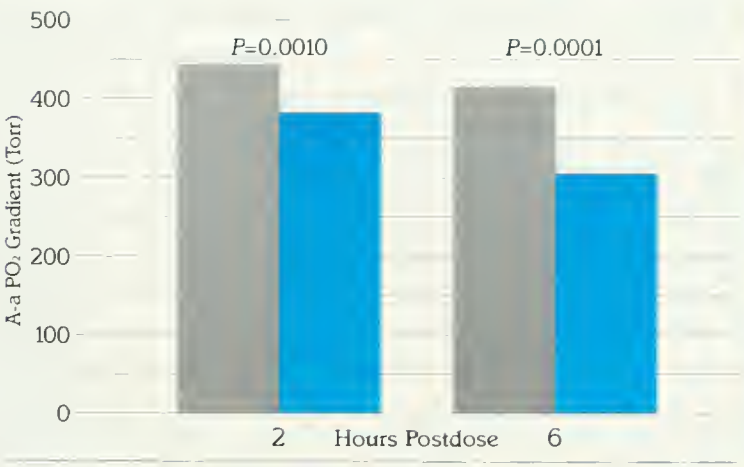
Rapid onset of action documented in rescue use[4]

Improvements in mean FiO_2 and mean alveolar-arterial PO_2 (A-a) gradient were present by 2 hours after dosing in middle-size babies (700-1350 grams). Improvements in mean airway pressures began sometime between 2 and 6 hours in middle-size babies. These improvements persisted for at least 7 days.

SIGNIFICANT IMPROVEMENTS IN SUPPLEMENTAL OXYGEN NEEDS AND VENTILATORY REQUIREMENTS SEEN IN MIDDLE-SIZE BABIES[4]







Air

EXOSURF Neonatal

All charts from Long[4]

Please see brief summary of full prescribing information on last pages of this advertisement.

Efficacy and impressive safety profile of EXOSURF® Neonatal™ confirmed in continued widespread use[6]

In North American controlled clinical trials, more than 2600 premature infants received EXOSURF Neonatal. Under the year-long Treatment IND, over 11,400 infants received EXOSURF Neonatal. In the six months following its release for marketing, EXOSURF Neonatal has been given to 10,000 infants in more than 750 hospitals.

There are no known infectious or immunologic risks associated with EXOSURF Neonatal use. In controlled clinical trials, adverse events were comparable to those of placebo, with the exception of apnea and pulmonary bleeding. Infants receiving EXOSURF Neonatal required less ventilatory support, possibly contributing to an increased incidence of apnea. Pulmonary bleeding occurred in 1% of control infants and 2% of treated infants in controlled trials. In the treatment IND, pulmonary bleeding was reported in 4% and mucous plugging at a rate of 3/1000. Pulmonary bleeding appears to be preventable with early diagnosis and appropriate treatment of patent ductus arteriosus.

One-year follow-up evaluated developmental outcomes[7]

Double-blind 1-year follow-up of more than 1450 infants enrolled in randomized trials showed that mental and motor scores appeared to be higher in tiny infants (<750 grams) as well as middle-size infants (750-1249 grams) who received Exosurf Neonatal.

Economic data analysis showed cost savings[8-10]

Three separate studies evaluated the economic impact of a single prophylactic dose of EXOSURF Neonatal, two-dose rescue treatment in 700- to 1350-gram infants, and two-dose rescue treatment during the neonatal period in infants weighing over 1350 grams. Results indicate that both prophylactic treatment and rescue treatment are cost-effective. Mean hospital charges were $6451 less for large infants receiving two-dose rescue treatment versus air in the first 28 days of life.[10]

As easy to use as it is effective

- **Easy to store and use** EXOSURF Neonatal may be stored at room temperature. Reconstituted suspension may be maintained refrigerated or at room temperature for up to 12 hours. Key items needed for administration are supplied in one kit.
- **Easy to administer** Each EXOSURF Neonatal dose is administered in two 2.5-mL/kg half-doses without interrupting mechanical ventilation.
- **Easy on infant** To assist the distribution of EXOSURF Neonatal in the lungs, the infant is simply turned from midline position to the right after the first half-dose, and from midline position to the left after the second half-dose.

References: 1. Bose C, Corbet A, Bose G, et al. Improved outcome at 28 days of age for very low birth weight infants treated with a single dose of a synthetic surfactant. *J Pediatr.* 1990;117:947-953. **2.** Corbet A, Bucciarelli R, Goldman S, et al. Decreased mortality in small premature infants treated at birth with a single dose of synthetic surfactant: a multi-center controlled trial. *J Pediatr.* 1991;118:277-284. **3.** Gerdes J, Cook L, Beaumont E, Corbet A, Long W. American EXOSURF Pediatric Groups I and II. Effects of three vs. one prophylactic doses of EXOSURF Neonatal in 700- to 1100-gram neonates. *Pediatr Res.* 1991;29(no. 4, pt 2):214A. Abstract. **4.** Long W, Thompson T, Sundell H, et al. Effects of two rescue doses of a synthetic surfactant on mortality rate and survival without bronchopulmonary dysplasia in 700- to 1350-gram infants with respiratory distress syndrome. *J Pediatr.* 1991;118:595-605. **5.** U.S. and Canadian EXOSURF Pediatric Study Groups. Effects of two rescue doses of EXOSURF® Pediatric in 1232 infants ≥1250 grams. *Pediatr Res.* 1990;27(no. 4, pt 2):320A Abstract. **6.** Data on file, Burroughs Wellcome Co., 1991. **7.** Walter D, McGuinness G, Bose C, et al. Double-blind one-year follow-up in 1450 infants randomized to EXOSURF® Neonatal or air. *Pediatr Res.* 1991;29(no. 4, pt 2):270A. Abstract. **8.** Sell M, Corbet A, Gong A, et al. Economic impact of a single prophylactic dose of EXOSURF® Neonatal in 700- to 1100-gram infants. *Pediatr Res.* 1991;29(no. 4, pt 2):265A. Abstract. **9.** Mammel M, Mullett M, Derleth D, et al. Economic impact of two rescue doses of EXOSURF® Neonatal in 700-1350 gram infants. *Pediatr Res.* 1991;29(no. 4, pt 2):260A. Abstract. **10.** Schumacher R, Burchfield D, Vaughan R, et al. Economic impact of two rescue doses of EXOSURF® Neonatal in ≥1350 gram infants. *Pediatr Res.* 1991;29(no. 4, pt 2):264A. Abstract.

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Wellcome

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EX-Y01228

EXOSURF® Neonatal™
(COLFOSCERIL PALMITATE, CETYL ALCOHOL, TYLOXAPOL)
For Intratracheal Suspension / 10 mL vials

PLEASE CONSULT FULL PRODUCT INFORMATION BEFORE PRESCRIBING

INDICATIONS AND USAGE. Exosurf Neonatal is indicated for

1. **Prophylactic** treatment of infants with birth weights of less than 1350 grams who are at risk of developing RDS (see PRECAUTIONS).
2. **Prophylactic** treatment of infants with birth weights greater than 1350 grams who have evidence of pulmonary immaturity and
3. **Rescue** treatment of infants who have developed RDS

CONTRAINDICATIONS. There are no known contraindications to treatment with Exosurf Neonatal.

WARNINGS: Intratracheal Administration Only: Exosurf Neonatal should be administered only by instillation into the trachea. **General**: The use of Exosurf Neonatal requires expert clinical care by experienced neonatologists and other clinicians who are accomplished at neonatal intubation and ventilatory management. Adequate personnel, facilities, equipment, and medications are required to optimize perinatal outcome in premature infants. Instillation of Exosurf Neonatal should be performed **only** by trained medical personnel experienced in airway and clinical management of unstable premature infants. Vigilant clinical attention should be given to all infants prior to, during, and after administration of Exosurf Neonatal. **Acute Effects**: Exosurf Neonatal can rapidly affect oxygenation and lung compliance. **Lung Compliance**: If chest expansion improves substantially after dosing, peak ventilator inspiratory pressures should be reduced immediately, without waiting for confirmation of respiratory improvement by blood gas assessment. Failure to reduce inspiratory ventilator pressures rapidly in such instances can result in lung overdistention and fatal pulmonary air leak. **Hyperoxia**: If the infant becomes pink and transcutaneous oxygen saturation is in excess of 95%, FiO_2 should be reduced in small but repeated steps (until saturation is 90 to 95%) without waiting for confirmation of elevated arterial pO_2 by blood gas assessment. Failure to reduce FiO_2 in such instances can result in hyperoxia. **Hypocarbia**: If arterial or transcutaneous CO_2 measurements are <30 torr, the ventilator rate should be reduced at once. Failure to reduce ventilator rates in such instances can result in marked hypocarbia, which is known to reduce brain blood flow. **Pulmonary Hemorrhage**: In the single study conducted in infants weighing <700 grams at birth, the incidence of pulmonary hemorrhage (10% vs 2% in the placebo group) was significantly increased in the Exosurf Neonatal group. None of the five studies involving infants with birth weights >700 grams showed a significant increase in pulmonary hemorrhage in the Exosurf Neonatal group. In a cross-study analysis of these five studies, pulmonary hemorrhage was reported for 1% (14/1420) of infants in the placebo group and 2% (27/1411) of infants in the Exosurf Neonatal group. Fatal pulmonary hemorrhage occurred in three infants, two in the Exosurf Neonatal group and one in the placebo group. Mortality from all causes among infants who developed pulmonary hemorrhage was 43% in the placebo group and 37% in the Exosurf Neonatal group. Pulmonary hemorrhage in both Exosurf Neonatal and placebo infants was more frequent in infants who were younger, smaller, male, or who had a patent ductus arteriosus. Pulmonary hemorrhage typically occurred in the first 2 days of life in both treatment groups. In more than 7700 infants in the open, uncontrolled study, pulmonary hemorrhage was reported in 4%, but fatal pulmonary hemorrhage was reported rarely (0.4%). In the controlled clinical studies, Exosurf Neonatal treated infants who received steroids more than 24 hours prior to delivery or indomethacin postnatally had a lower rate of pulmonary hemorrhage than other Exosurf Neonatal treated infants. Attention should be paid to early and aggressive diagnosis and treatment (unless contraindicated) of patent ductus arteriosus during the first 2 days of life (while the ductus arteriosus is often clinically silent). Other potentially protective measures include attempting to decrease FiO_2 preferentially over ventilator pressures during the first 24 to 48 hours after dosing, and attempting to decrease PEEP minimally for at least 48 hours after dosing. **Mucous Plugs**: Infants whose ventilation becomes markedly impaired during or shortly after dosing may have mucous plugging of the endotracheal tube, particularly if pulmonary secretions were prominent prior to drug administration. Suctioning of all infants prior to dosing may lessen the chance of mucous plugs obstructing the endotracheal tube. If endotracheal tube obstruction from such plugs is suspected, and suctioning is unsuccessful in removing the obstruction, the blocked endotracheal tube should be replaced immediately.

PRECAUTIONS

General: In the controlled clinical studies, infants known prenatally or postnatally to have major congenital anomalies, or who were suspected of having congenital infection, were excluded from entry. However, these disorders cannot be recognized early in life in all cases, and a few infants with these conditions were entered. The benefits of Exosurf Neonatal in the affected infants who received drug appeared to be similar to the benefits observed in infants without anomalies or occult infection. **Prophylactic Treatment–Infants <700 Grams**: In infants weighing 500 to 700 grams, a single prophylactic dose of Exosurf Neonatal significantly improved FiO_2 and ventilator settings, reduced pneumothorax, and reduced death from RDS, but increased pulmonary hemorrhage (see WARNINGS). Overall mortality did not differ significantly between the placebo and Exosurf Neonatal groups (see Table 1 in full product information). Data on multiple doses in infants in this weight class are not yet available. Accordingly, clinicians should carefully evaluate the potential risks and benefits of Exosurf Neonatal administration in these infants. **Rescue Treatment–Number of Doses**: A small number of infants with RDS have received more than two doses of Exosurf Neonatal as rescue treatment. Definitive data on the safety and efficacy of these additional doses are not available. **Carcinogenesis, Mutagenesis, Impairment of Fertility**: Exosurf Neonatal at concentrations up to 10 000 µg/plate was not mutagenic in the Ames Salmonella assay. Long-term studies have not been performed in animals to evaluate the carcinogenic potential of Exosurf Neonatal. The effects of Exosurf Neonatal on fertility have not been studied.

ADVERSE REACTIONS

General: Premature birth is associated with a high incidence of morbidity and mortality. Despite significant reductions in overall mortality associated with Exosurf Neonatal, some infants who received Exosurf Neonatal developed severe complications and either survived with permanent handicaps or died.

In controlled clinical studies evaluating the safety and efficacy of Exosurf Neonatal, numerous safety assessments were made. In infants receiving Exosurf Neonatal, pulmonary hemorrhage, apnea and use of methylxanthines were increased. A number of other adverse events were significantly reduced in the Exosurf Neonatal group, particularly various forms of pulmonary air leak and use of pancuronium. Tables 3 and 4 summarize the results of the major safety evaluations from the controlled clinical studies.

Table 3: Safety Assessments[a]—Prophylactic Treatment

Number of Doses / Birth Weight Range	Single Dose 500 to 700 grams		Single Dose 700 to 1350 grams		Single Dose 700 to 1100 grams		1 vs 3 Doses 700 to 1100 grams	
Treatment Group / Number of Infants	Placebo (Air) N=108	EXOSURF N=107	Placebo (Air) N=193	EXOSURF N=192	Placebo (Air) N=222	EXOSURF N=224	EXOSURF 1 Dose N=356	EXOSURF 3 Doses N=360
	% of Infants		% of Infants		% of Infants		% of Infants	
Intraventricular Hemorrhage (IVH)								
Overall	51	57	31	27	36	36	38	35
Severe IVH	26	25	10	8	13	14	9	9
Pulmonary Air Leak (PAL)								
Overall	52	48	16	11	32	25	29	27
Pneumothorax	23	10*	5	6	19	11*	14	12
Pneumopericardium	1	4	2	0	<1	1	1	1
Pneumomediastinum	2	1	2	3	7	1**	3	2
Pulmonary Interstitial Emphysema	43	44	13	7*	26	20	23	22
Death from PAL	4	6	<1	<1	2	1	2	1
Patent Ductus Arteriosus	49	53	66	70	50	55	59	57
Necrotizing Enterocolitis	2	4	11	13	3	4	6	2*
Pulmonary Hemorrhage	2	10**	2	4	1	4	4	6
Congenital Pneumonia	4	4	2	4	2	2	1	1
Nosocomial Pneumonia	10	10	2	4	4	7	14	15
Non-Pulmonary Infections	33	35	34	39	28	29	35	34
Sepsis	30	34	30	34	23	24	30	27
Death From Sepsis	4	4	3	3	1	2	3	2
Meningitis	4	6	3	1	2	3	1	2
Other Infections	7	4	5	3	6	10	10	11
Major Anomalies	3	1	2	4	7	4	4	4

Table 3: Safety Assessments[a]—Prophylactic Treatment (Cont.)

Number of Doses / Birth Weight Range	Single Dose 500 to 700 grams		Single Dose 700 to 1350 grams		Single Dose 700 to 1100 grams		1 vs 3 Doses 700 to 1100 grams	
Treatment Group / Number of Infants	Placebo (Air) N=108	EXOSURF N=107	Placebo (Air) N=193	EXOSURF N=192	Placebo (Air) N=222	EXOSURF N=224	EXOSURF 1 Dose N=356	EXOSURF 3 Doses N=360
	% of Infants		% of Infants		% of Infants		% of Infants	
Hypotension	70	77	52	47	59	62	54	50
Hyperbilirubinemia	22	21	63	61	27	31	20	21
Exchange Transfusion	4	3	1	2	2	2	3	1
Thrombocytopenia[b]	21	25	not available		9	8	12	10
Persistent Fetal Circulation	0	1	1	1	0	2*	1	<1
Seizures	11	8	2	2	11	9	6	5
Apnea	34	33	76	73	55	65*	62	68
Drug Therapy								
Antibiotics	96	99	98	96	96	99	>99	99
Diuretics	55	60	39	37	59	63	64	65
Anticonvulsants	14	18	23	24	20	16	9	8
Inotropes	46	40	20	20	26	20	26	27
Sedatives	62	71	65	64	63	57	52	52
Pancuronium	19	11	22	14*	19	13*	15	11
Methylxanthines	38	43	77	77	61	72*	75	82*

[a] All parameters were examined with "as-treated" analyses. * p<0.05. ** p<0.01.
[b] Thrombocytopenia requiring platelet transfusion.

Table 4: Safety Assessments[a]—Rescue Treatment

Number of Doses / Birth Weight Range	Two Doses 700 to 1350 grams		Two Doses 1250 grams and above	
Treatment Group / Number of Infants	Placebo (Air) N=213	EXOSURF N=206	Placebo (Air) N=622	EXOSURF N=615
	% of Infants		% of Infants	
Intraventricular Hemorrhage (IVH)				
Overall	48	52	23	18*
Severe IVH	13	9	5	4
Pulmonary Air Leak (PAL)				
Overall	54	34***	30	18***
Pneumothorax	29	20*	20	10***
Pneumopericardium	4	1	1	2
Pneumomediastinum	8	4	5	2**
Pulmonary Interstitial Emphysema	48	25***	24	13***
Death from PAL	7	3	<1	1
Patent Ductus Arteriosus	66	57	54	45*
Necrotizing Enterocolitis	3	3	1	2
Pulmonary Hemorrhage	3	1	<1	1
Congenital Pneumonia	2	3	2	2
Nosocomial Pneumonia	5	7	2	2
Non-Pulmonary Infections	19	22	13	13
Sepsis	15	17	8	8
Death From Sepsis	<1	<1	1	<1
Meningitis	1	<1	1	<1*
Other Infections	5	8	5	6
Major Anomalies	3	3	4	4
Hypotension	62	57	50	39**
Hyperbilirubinemia	17	19	12	10
Exchange Transfusion	3	4	1	2
Thrombocytopenia[b]	10	11	4	<1**
Persistent Fetal Circulation	1	1	6	2**
Seizures	10	10	6	3*
Apnea	48	65**	37	44*
Drug Therapy				
Antibiotics	100	99	98	98
Diuretics	60	65	45	34***
Anticonvulsants	17	17	10	5**
Inotropes	36	31	27	16***
Sedatives	72	68	76	64***
Pancuronium	34	17**	33	15***
Methylxanthines	62	74**	49	53

[a] All parameters were examined with "as-treated" analyses. * p<0.05. ** p<0.01. *** p<0.001.
[b] Thrombocytopenia requiring platelet transfusion.

Pulmonary Hemorrhage. See WARNINGS. **Abnormal Laboratory Values**. Abnormal laboratory values are common in critically ill, mechanically ventilated, premature infants. A higher incidence of abnormal laboratory values in the Exosurf Neonatal group was not reported. **Events During Dosing**. Data on events during dosing are available from more than 8800 infants in the open, uncontrolled clinical study (Table 5).

Table 5. Events During Dosing in the Open, Uncontrolled Study[a]

Treatment Type / Number of Infants	Prophylactic Treatment N=1127	Rescue Treatment N=7711
	% of Infants	% of Infants
Reflux of Exosurf	20	31
Drop in O_2 saturation (≥ 20%)	6	22
Rise in O_2 saturation (≥ 10%)	5	6
Drop in transcutaneous pO_2 (≥ 20 mm Hg)	1	8
Rise in transcutaneous pO_2 (≥ 20 mm Hg)	2	5
Drop in transcutaneous pCO_2 (≥ 20 mm Hg)	<1	1
Rise in transcutaneous pCO_2 (≥ 20 mm Hg)	1	3
Bradycardia (<60 beats/min)	1	3
Tachycardia (>200 beats/min)	<1	<1
Gagging	1	5
Mucous Plugs	<1	<1

[a] Infants may have experienced more than one event.
Investigators were prohibited from adjusting FiO_2 and/or ventilator settings during dosing unless significant clinical deterioration occurred.

Reflux. Reflux of Exosurf Neonatal into the endotracheal tube during dosing has been observed and may be associated with rapid drug administration. If reflux occurs, drug administration should be halted and, if necessary, peak inspiratory pressure on the ventilator should be increased by 4 to 5 cm H_2O until the endotracheal tube clears. ***>20% Drop in Transcutaneous Oxygen Saturation***. If transcutaneous oxygen saturation declines during dosing, drug administration should be halted and, if necessary, peak inspiratory pressure on the ventilator should be increased by 4 to 5 cm H_2O for 1 to 2 minutes. In addition, increases of FiO_2 may be required for 1 to 2 minutes. ***Mucous Plugs***. See WARNINGS.

DOSAGE AND ADMINISTRATION. See full product information.

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significant differences in baseline FEV_1 after exposure to ozone but PC_{15} was significantly reduced when allergen was preceded by ozone, inhalation: The mean PC_{15} after air was 0.013 ([SD] 0.017) mg/mL compared with 0.0056 (0.0062) mg/mL after ozone ($p = 0.042$). Thus, low ozone concentrations, similar to those commonly occurring in urban areas, can increase the bronchial responsiveness to allergen in atopic asthmatic subjects. This effect does not seem to be the result of changes in baseline airway function.

Diagnosis of Nosocomial Pneumonia in Mechanically Ventilated Patients: Comparison of a Plugged Telescoping Catheter with the Protected Specimen Brush—LH Pham, C Brun-Buisson, P Legrand, A Rauss, F Verra, L Brochard, et al. Am Rev Respir Dis 1991;143:1055.

Protected samples of lower respiratory tract secretions processed by quantitative culture techniques are recommended to diagnose nosocomial bacterial pneumonia in intubated, mechanically ventilated patients. To evaluate the accuracy of a simple and inexpensive sampling device in this setting, we compared quantitative cultures of paired single-sheathed plugged telescoping catheter (PTC) and protected specimen brush (PSB) samples in 55 patients during 78 suspected episodes of nosocomial pneumonia. PTC and PSB samples were taken in randomized order, and patients were also randomized to have PTC samples taken "blindly" or via a fiberoptic bronchoscope. Fifteen PSB and 27 PTC samples were culture positive ($\geq 10^3$ cfu/mL). The two sampling procedures gave similar results in 58 (74%) episodes. A major discrepancy occurred in 20 episodes, including six false-negatives of PSB in episodes of proved pneumonia, four possible false-positives of PSB, and 10 possible false-positives of PTC (three of which rapidly evolved toward overt pneumonia). The sensitivity and specificity of PTC were 100 and 82.2%, and those of PSB were 64.7 and 93.5%, respectively. Blinded or directed PTC samples had similar concordance with PSB samples taken via bronchoscopy. We conclude that PTC is at least as accurate as PSB in the bacteriologic diagnosis of nosocomial pneumonia in intubated patients, and that its use can result in substantial cost savings, especially when fiberoptic bronchoscopy is not otherwise indicated.

Aerosol Therapy Implications of Particle Deposition Patterns in Simulated Human Airways—TB Martonen. J Aerosol Med 1991;4:25.

The efficacy of inhalation therapy may be improved by the selective deposition of aerosolized medicines, by explicitly targeting and delivering drugs to prescribed lung sites. This may be accomplished by controlling particle sizes and regulating breathing patterns. Here, the deposition patterns of 1.9-10.6 μm mass median aerodynamic diameter test aerosols, mapped in surrogate respiratory tracts consisting of replica laryngeal casts and fabricated tracheobronchial models, are analyzed. Configurations of the laryngeal casts corresponded to inspiratory flowrates of 15, 30, and 60 $L \cdot min^{-1}$. Particles were preferentially deposited at bifurcations, specifically at carinal ridges. Evidence from other investigations indicates that mucociliary clearance may be impeded at such sites in vivo. When these two effects, enhanced deposition and impaired clearance, are coupled, the findings suggest that epithelial cells and receptors at airway branching sites may receive concentrated doses of inhaled pharmacological agents. To focus the effects of airborne drugs the lung should, therefore, be considered as a serial network of Y-shaped bifurcation units. The findings have important implications to current aerosol therapy using metered dose inhalers and nebulizers, and future protocols, including: (1) the treatment of bronchogenic carcinomas because malignant tumors have a predilection for upper airway bifurcations; and (2) lung diseases related to the afferent nervous system since components of neural pathways frequent such locations.

Symptomatic Benefit of Supplemental Oxygen in Hypoxemic Patients with Chronic Lung Disease—C R Swinburn, H Mould, TN Stone, PA Corris, GJ Gibson. Am Rev Respir Dis 1991;143:913.

We have compared the symptomatic benefit of air and oxygen at rest in hypoxemic patients with chronic obstructive airway disease (COAD) or interstitial lung disease (ILD). A total of 12 severely disabled patients with COAD (mean ± SEM, P_{aO_2} 50.3 ± 3.7 torr) and 10 with ILD (P_{aO_2}, 48.0 ± 3.1 torr) received 28% oxygen and air by Venturi face mask, each gas on two occasions, in a double-blind randomized fashion. S_{aO_2} increased ($p < 0.01$) in both groups during oxygen breathing: COAD, 85.1 ± 2.3% versus 93.1 ± 1.4%; ILD, 85.5 ± 1.7% versus 94.7 ± 0.9%. The patients with COAD stated that air helped their breathing on 15 of 24 occasions and that oxygen helped on 22 of 24 occasions ($p < 0.05$). In the patients with ILD the values were 6 of 20 and 13 of 20 occasions, respectively ($p < 0.05$). In both groups of patients the severity of breathlessness recorded on a 100-mm visual analog scale was significantly ($p < 0.05$) lower during oxygen breathing: COAD, 29.6 ± 4.5 versus 45.6 ± 6.0; ILD, 30.2 ± 5.1

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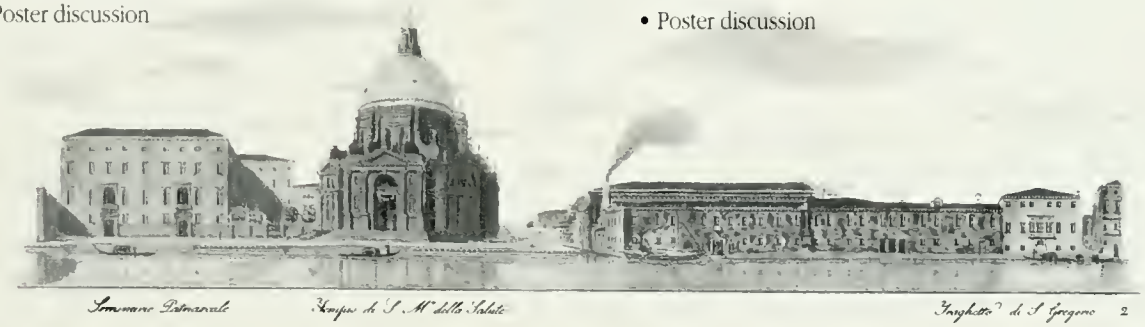

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versus 48.1 ± 4.4. Ventilation measured by magnetometers was significantly lower during oxygen breathing in the patients with COAD (8.2 ± 1.0 versus 9.3 ± 1.1 L/min; $p < 0.05$), but the difference between oxygen and air in patients with ILD was not statistically significant (9.3 ± 1.3 versus 11.2 ± 1.6 L/min; $p > 0.05$). We conclude that hypoxemic and breathless patients with COAD or ILD benefit symptomatically at rest from supplemental oxygen and to a greater extent than from air delivered in similar fashion via face mask.

Oropharyngeal Decontamination Decreases Incidence of Ventilator-Associated Pneumonia: A Randomized, Placebo-Controlled, Double-Blind Clinical Trial—J Pugin, R Auckenthaler, DP Lew, PM Suter. JAMA 1991;265:2704.

Secondary pneumonia in patients requiring mechanical ventilation has a high morbidity and mortality. Diagnosis is difficult and treatment failure common; therefore, preventive measures are important. In a double-blind, placebo-controlled trial, we evaluated selective decontamination of the oropharynx with polymyxin B sulfate, neomycin sulfate, and vancomycin hydrochloride (PNV) in 52 patients requiring mechanical ventilation during a 3- to 34-day period (mean, 10 days). Either PNV or placebo was administered six times daily in the oropharynx. During the first 12 days of intubation, tracheobronchial colonization by gram-negative bacteria and *Staphylococcus aureus*, as well as pneumonia, occurred less frequently in the PNV than in the placebo group (16% vs 78%; $p < 0.0001$). Hospital mortality was not different, but systemic antibiotics were prescribed less often in the PNV group and no resistant microorganism emerged. In these critically ill patients, topical oropharyngeal antibiotic application lowered the rate of ventilator-associated pneumonia by a factor of 5, probably by interrupting the stomach-to-trachea route of infection, and decreased the requirement for intravenous antibiotics.

Nasal Continuous Positive Airway Pressure Facilitates Respiratory Muscle Function during Sleep in Severe Chronic Obstructive Pulmonary Disease—BJ Petrof, RJ Kimoff, RD Levy, MG Cosio, SB Gottfried. Am Rev Respir Dis 1991; 143:928.

Patients with chronic respiratory insufficiency due to severe chronic obstructive pulmonary disease (COPD) and presumed respiratory muscle fatigue may benefit from therapeutic maneuvers aimed at reducing the magnitude of inspiratory muscle effort. Recent work has demonstrated that continuous positive airway pres-

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INDICATIONS AND USAGE

SURVANTA is indicated for prevention and treatment ("rescue") of Respiratory Distress Syndrome (RDS) (hyaline membrane disease) in premature infants. SURVANTA significantly reduces the incidence of RDS, mortality due to RDS and air leak complications.

Prevention

In premature infants less than 1250 g birth weight or with evidence of surfactant deficiency, give SURVANTA as soon as possible, preferably within 15 minutes of birth.

Rescue

To treat infants with RDS confirmed by x-ray and requiring mechanical ventilation, give SURVANTA as soon as possible, preferably by 8 hours of age.

CONTRAINDICATIONS

None known.

WARNINGS

SURVANTA is intended for intratracheal use only.

SURVANTA CAN RAPIDLY AFFECT OXYGENATION AND LUNG COMPLIANCE. Therefore, its use should be restricted to a highly supervised clinical setting with immediate availability of clinicians experienced with intubation, ventilator management, and general care of premature infants. Infants receiving SURVANTA should be frequently monitored with arterial or transcutaneous measurement of systemic oxygen and carbon dioxide.

DURING THE DOSING PROCEDURE, TRANSIENT EPISODES OF BRADYCARDIA AND DECREASED OXYGEN SATURATION HAVE BEEN REPORTED. If these occur, stop the dosing procedure and initiate appropriate measures to alleviate the condition. After stabilization, resume the dosing procedure.

PRECAUTIONS

General

Rales and moist breath sounds can occur transiently after administration. Endotracheal suctioning or other remedial action is not necessary unless clear-cut signs of airway obstruction are present.

Increased probability of post-treatment nosocomial sepsis in SURVANTA-treated infants was observed in the controlled clinical trials (Table 3). The increased risk for sepsis among SURVANTA-treated infants was not associated with increased mortality among these infants. The causative organisms were similar in treated and control infants. There was no significant difference between groups in the rate of post-treatment infections other than sepsis.

Use of SURVANTA in infants less than 600 g birth weight or greater than 1750 g birth weight has not been evaluated in controlled trials. There is no controlled experience with use of SURVANTA in conjunction with experimental therapies for RDS (eg, high-frequency ventilation or extracorporeal membrane oxygenation).

No information is available on the effects of doses other than 100 mg phospholipids/kg, more than four doses, dosing more frequently than every 6 hours, or administration after 48 hours of age.

Carcinogenesis, Mutagenesis, Impairment of Fertility

Reproduction studies in animals have not been completed. Mutagenicity studies were negative. Carcinogenicity studies have not been performed with SURVANTA.

ADVERSE REACTIONS

The most commonly reported adverse experiences were associated with the dosing procedure. In the multiple-dose controlled clinical trials, transient bradycardia occurred with 11.9% of doses. Oxygen desaturation occurred with 9.8% of doses.

Other reactions during the dosing procedure occurred with fewer than 1% of doses and included endotracheal tube reflux, pallor, vasoconstriction, hypotension, endotracheal tube blockage, hypertension, hypocarbia, hypercarbia, and apnea. No deaths occurred during the dosing procedure, and all reactions resolved with symptomatic treatment.

The occurrence of concurrent illnesses common in premature infants was evaluated in the controlled trials. The rates in all controlled studies are in Table 3.

TABLE 3

	All Controlled Studies		
Concurrent Event	SURVANTA (%)	Control (%)	*P* Value[a]
Patent ductus arteriosus	46.9	47.1	0.814
Intracranial hemorrhage	48.1	45.2	0.241
Severe intracranial hemorrhage	24.1	23.3	0.693
Pulmonary air leaks	10.9	24.7	< 0.001
Pulmonary interstitial emphysema	20.2	38.4	< 0.001
Necrotizing enterocolitis	6.1	5.3	0.427
Apnea	65.4	59.6	0.283
Severe apnea	46.1	42.5	0.114
Post-treatment sepsis	20.7	16.1	0.019
Post-treatment infection	10.2	9.1	0.345
Pulmonary hemorrhage	7.2	5.3	0.166

[a]*P*-value comparing groups in controlled studies

When all controlled studies were pooled, there was no difference in intracranial hemorrhage. However, in one of the single-dose rescue studies and one of the multiple-dose prevention studies, the rate of intracranial hemorrhage was significantly higher in SURVANTA patients than control patients (63.3% *v* 30.8%, $P = 0.001$, and 48.8% *v* 34.2%, $P = 0.047$, respectively). The rate in a Treatment INO involving approximately 4400 infants was lower than in the controlled trials.

In the controlled clinical trials, there was no effect of SURVANTA on results of common laboratory tests: white blood cell count and serum sodium, potassium, bilirubin, creatinine.

More than 3700 pretreatment and post-treatment serum samples were tested by Western Blot immunoassay for antibodies to surfactant-associated proteins SP-B and SP-C. No IgG or IgM antibodies were detected.

Several other complications are known to occur in premature infants. The following conditions were reported in the controlled clinical studies. The rates of the complications were not different in treated and control infants, and none of the complications were attributed to SURVANTA.

Respiratory: lung consolidation, blood from the endotracheal tube, deterioration after weaning, respiratory decompensation, subglottic stenosis, paralyzed diaphragm, respiratory failure.

Cardiovascular: hypotension, hypertension, tachycardia, ventricular tachycardia, aortic thrombosis, cardiac failure, cardiorespiratory arrest, increased apical pulse, persistent fetal circulation, air embolism, total anomalous pulmonary venous return.

Gastrointestinal: abdominal distention, hemorrhage, intestinal perforations, volvulus, bowel infarct, feeding intolerance, hepatic failure, stress ulcer.

Renal: renal failure, hematuria.

Hematologic: coagulopathy, thrombocytopenia, disseminated intravascular coagulation.

Central Nervous System: seizures.

Endocrine/Metabolic: adrenal hemorrhage, inappropriate ADH secretion, hyperphosphatemia.

Musculoskeletal: inguinal hernia.

Systemic: fever, deterioration.

Follow-Up Evaluations

To date, no long-term complications or sequelae of SURVANTA therapy have been found.

Single-Dose Studies

Six-month adjusted-age follow-up evaluations of 232 infants (115 treated) demonstrated no clinically important differences between treatment groups in pulmonary and neurologic sequelae, incidence or severity of retinopathy of prematurity, rehospitalizations, growth, or allergic manifestations.

Multiple-Dose Studies

Six-month adjusted age follow-up evaluations have not been completed. Preliminarily, in 605 (333 treated) of 916 surviving infants, there are trends for decreased cerebral palsy and need for supplemental oxygen in SURVANTA infants. Wheezing at the time of examination tended to be more frequent among SURVANTA infants, although there was no difference in bronchodilator therapy.

Twelve-month follow-up data from the multiple-dose studies have been completed in 328 (171 treated) of 909 surviving infants. To date no significant differences between treatments have been found, although there is a trend toward less wheezing in SURVANTA infants in contrast to the six month results.

OVERDOSAGE

Overdosage with SURVANTA has not been reported. Based on animal data, overdosage might result in acute airway obstruction. Treatment should be symptomatic and supportive.

Rales and moist breath sounds can transiently occur after SURVANTA is given, and do not indicate overdosage. Endotracheal suctioning or other remedial action is not required unless clear-cut signs of airway obstruction are present.

HOW SUPPLIED

SURVANTA (beractant) Intratracheal Suspension is supplied in single-use glass vials containing 8 mL of SURVANTA (NDC 0074-1040-08). Each milliliter contains 25 mg of phospholipids (200 mg phospholipids/8 mL) suspended in 0.9% sodium chloride solution. The color is off-white to light brown.

Store unopened vials at refrigeration temperature (2-8°C). Protect from light. Store vials in carton until ready for use. Vials are for single use only. Upon opening, discard unused drug.

June, 1991

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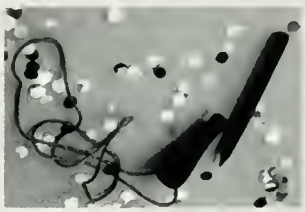

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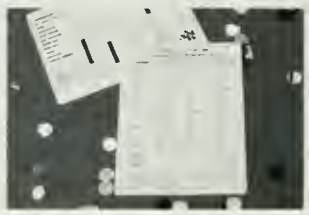

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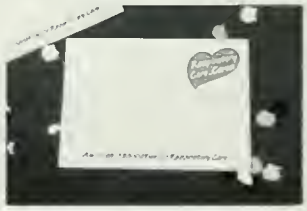

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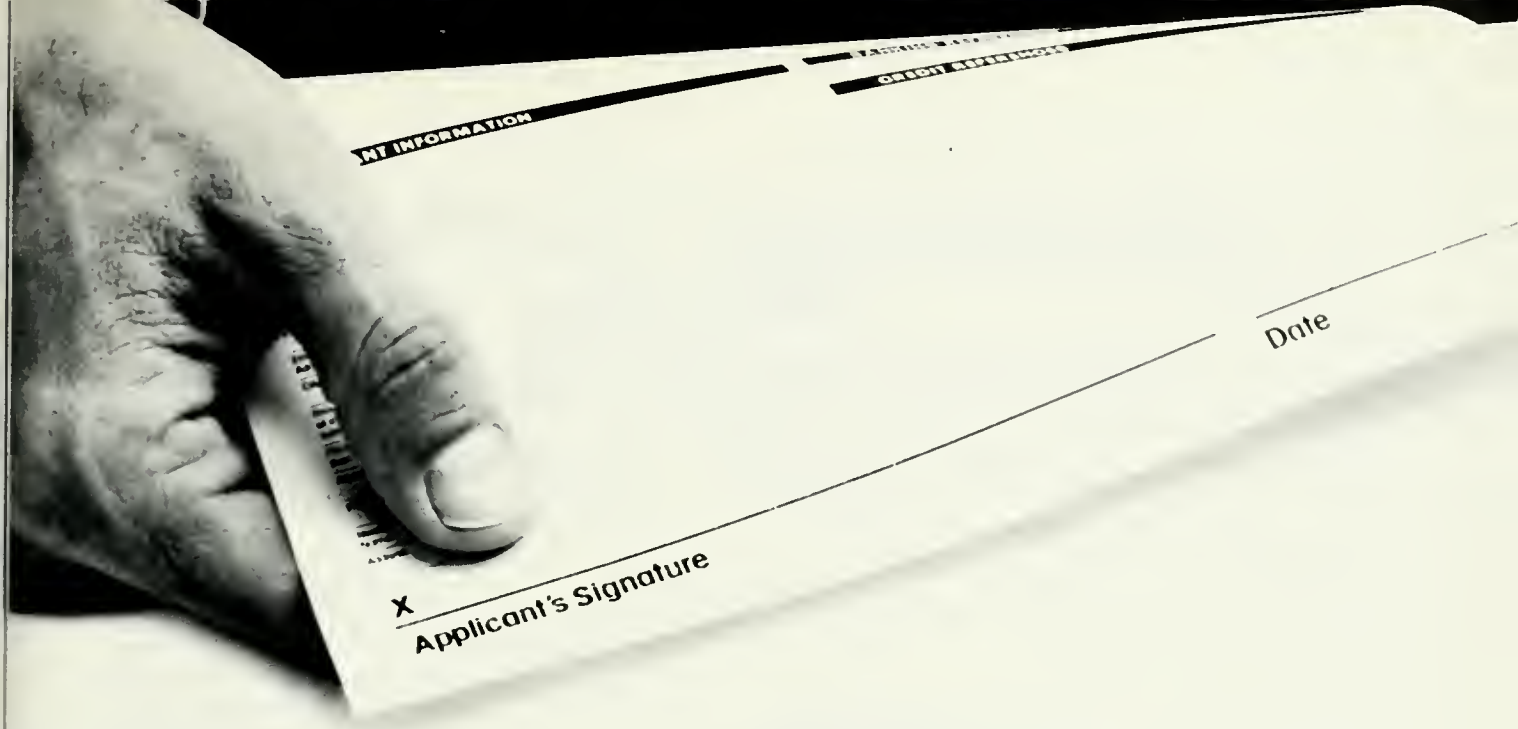

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sure (CPAP) can significantly reduce inspiratory effort and work of breathing in COPD patients with acute respiratory failure. Accordingly, it was reasoned that prolonged CPAP administration may similarly reduce the work of breathing in stable COPD patients with chronic respiratory insufficiency, thereby allowing recovery from respiratory muscle fatigue. The purpose of this study was to determine the feasibility of employing nasal CPAP during sleep as a means of implementing this approach to reducing inspiratory muscle effort in such patients. Standard polysomnographic parameters were recorded during nocturnal administration of nasal CPAP in eight stable patients with severe COPD (FEV_1 = 26.7 ± 3.9% of predicted). Esophageal pressure, diaphragmatic (EMGdi) and parasternal intercostal (EMGic) electromyographic activity, arterial oxyhemoglobin saturation (S_{aO_2}), and transcutaneous P_{tcCO_2} were also measured. Breathing pattern was determined by respiratory inductive plethysmography. In each patient an optimum level of nasal CPAP could be determined that produced consistent reductions in indices of inspiratory muscle effort without changing tidal volume or breathing frequency. Highly significant reductions in the tidal excursions of esophageal pressure and the pressure-time integral for the inspiratory muscles occurred at the optimum CPAP level in all patients. EMGdi and EMGic were similarly reduced. S_{aO_2} and P_{tcCO_2} were unaffected by CPAP. These results indicate that nasal CPAP can effectively reduce inspiratory muscle effort during sleep in patients with severe COPD. Future work is required to determine whether long-term nasal CPAP administration can sufficiently reduce inspiratory effort to produce sustained improvement in muscle function in patients with severe COPD and presumed chronic respiratory muscle fatigue.

Home Care Cost-Effectiveness for Respiratory Technology-Dependent Children—Al Fields, A Rosenblatt, MM Pollack, J Kaufman. AJDC 1991;145:729.

We evaluated home care costs and the cost-effectiveness of home care vs alternative institutional care for respiratory technology-dependent children in a Medicaid Model Waiver Program. "Cost-savings" was measured as the difference between the established Medicaid reimbursable charges to enact an individualized care plan at a long-term care institution and the actual Medicaid reimbursements for home care. Ten patients—six dependent on mechanical ventilation and four with a tracheostomy who were receiving oxygen—were included in the analysis. The mean (± SD) annual home care costs were $109,836 ± $20,781 for ventilator-dependent children and $63,650 ± 12,350 for oxygen-dependent patients with a tracheostomy, representing annual savings of approximately $79,000 per patient and $83,000 per patient, respectively. The largest portion of home care reimbursements was for nursing care, accounting for 69.0% and 59.0% of the two patient groups. The full program (50 patients) has the potential for a savings of $4 million per year.

Relationship between Response to Inhaled Salbutamol and Methacholine Bronchial Provocation in Children with Suspected Asthma—H Bibi, M Montgomery, H Pasterkamp, V Chernick. Pediatr Pulmonol 1991;10:244.

Fifty children (27 females, 23 males) ages 6-15 years who were referred for evaluation of suspected asthma had baseline FEV_1 and FEF_{25-75} of ≥ 80% and FEF_{50} ≥ 70% of predicted values. All had these tests repeated on the same day, after inhaling salbutamol. On a subsequent day PC_{20} (methacholine) was determined as an index of bronchial hyperreactivity (BH). Fourteen age-matched healthy children (6 females, 8 males) were studied in a similar manner. There was no significant relationship between the PC_{20} and the change in FEF_{25-75} or FEF_{50} following salbutamol. There was a negative correlation between the initial FEV_1 (% predicted) and the percent change in FEV_1 following salbutamol ($p < 0.01$). An increase in FEV_1 of > 6% occurred in 7/12 (58.3%) patients with PC_{20} ≤ 0.25 mg/mL (Group I); in 7/24 (29.2%) patients with PC_{20} = 0.26-2.0 mg/mL (Group II); in only 1/14 (7.1%) patients with PC_{20} > 2.1 mg/mL (Group III) and in none of those asymptomatic (control) children with PC_{20} > 8.0 mg/mL (Group IV). All subjects who had a change in FEV_1 > 6% after salbutamol had a PC_{20} < 8 mg/mL and this test detected the majority of patients with severe BH. However, although the sensitivity of the test was 100%, the predictive value was only 36%. We conclude that in the presence of a normal baseline FEV_1 a change of > 6% following salbutamol inhalation is indicative of bronchial hyperreactivity.

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Professional Literacy

Not long ago, a professional colleague and I who had known each other for 25 years were engaged in a casual conversation covering a host of topics. At one point he blithely mentioned that he no longer bothered to read the journal RESPIRATORY CARE—implying that he just threw it out like so much junk mail! While I tried not to overtly react to this startling declaration, it left me deeply troubled, especially as I had always considered this man to be very bright, indeed. (Perhaps he considers himself so bright that he doesn't need continuing education.)

In my youth, the term "functional illiterate" was unknown. Ever since the onset of the Industrial Revolution, each successive generation in Western society had striven to surpass the previous one in intellectual curiosity, development, and attainment. That trend apparently has been curtailed in recent times because today, as this century closes, functional illiteracy is recognized as a major social and economic problem in our culture. For example, I have heard that the instructions for the operation of some major military hardware are provided in comic-book form because written instructions might not be understood by the soldiers who operate those devices. That is functional illiteracy in a frightening and uncomical setting. In the civilian setting, business and industry have expressed deep concern that job-seekers these days have poorer language and arithmetic skills than in former times—even those applicants with college degrees.

Why is this? What happened? Can it be that a resource-rich, free society provides enough of the stuff of survival that literacy is not deemed necessary? If this is the case, who has made this decision, whether consciously or not? Could it be the education system? Some years ago, Philip Kittredge, then Editor of this journal, polled educators at our Association's annual convention, asking them whether they assigned articles in RESPIRATORY CARE to be read and discussed by their advanced students. Some educators replied that they had tried to do this, but the students had not wanted to read such material. And those educators had acquiesced to their students' lack of desire!

Last year, in these pages, Mr Kittredge glanced over our collective shoulders to provide us with a look at the first 35 years in the life of *our* journal.[1] In doing so, he succinctly recalled the history of our meager beginnings and those steps that had led to our professional maturation, as reflected *in* RESPIRATORY CARE.

In 1987, Journal Editor Pat Brougher announced that henceforth RESPIRATORY CARE would be open to education and management articles.[2] To my mind, this step heralded the discipline's new level of self-esteem, representing a professional growth beyond pure clinical practice. It meant that thereafter, additional literary resources would be there for those who would avail themselves of them.

In my more than 30 years of clinical practice, the Journal has been the *one* tool always available to me in the constant struggle to remain professionally contemporary. By way of illustration, recently it became necessary for me to have a working knowledge of adult sleep apnea. To gain this knowledge, I did not have to leave my office. I was able to turn to Mishoe's 1987 article on the subject,[3] with its wonderfully elaborate list of references. This authoritative overview of the subject provided me with the details I needed to speak and act as an informed professional. Thank goodness I had not thrown out that 1987 journal issue as so much junk mail!

The 2,481 references cited in articles in RESPIRATORY CARE last year are, of themselves, worth the subscription price! They were used by 186 authors to buttress 130 original articles, letters, points of view, and special and feature articles.

Volume 35 (1990) also showcased 237 display advertisements (they can be educational), 53 new products, 21 product evaluations, 21 reviews of books and software, as well as 94 Open Forum abstracts. When the 211 abstracts from other sources are added, an interested respiratory care practitioner, or a pulmonary physician for that matter, would be hard-pressed to find an area not represented in this rich and tasty literary broth.

I wonder whether, like American society as a whole, we practitioners now have so many resources available to us (eg, computerized ventilators) that professional literacy is no longer considered a criterion for professional survival. How many of our academic and continuing educators today are making use of RESPIRATORY CARE in their work?

I find it both alarming and philosophically repugnant that the respiratory care practitioner licensing act of my own state, Texas, denies, for purposes of continuing education, the value of contemporary knowledge directly gleaned from original work appearing in the literature. Should not the State of Texas, and other states with similar laws, be indicted for perpetuating professional illiteracy? I certainly hope not. However, there is my longstanding acquaintance mentioned at the beginning of this editorial, who is sanctioned by our state licensing act to nap through 6 hours of perhaps unoriginal, uninspired, albeit 'accredited' lectures each year, with the assurance that he can be this lazy and still retain his practitioner's license. The state is licensing not only practice, but professional illiteracy

But then, there is this journal. In its 36th year, a handful of inspired researchers/authors and a small, dedicated journal staff still fill the udder of professional sustenance to suckle those of us who are interested.

Robert R Weilacher RRT
Coordinator
Pulmonary Rehabilitation
Memorial Hospital
Palestine, Texas

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Determination of Ventilatory Reserve in Mechanically Ventilated Patients: A Comparison of Techniques

Robert M Kacmarek PhD RRT, Kevin Foley RRT, Penny Cheever RRT, and David Romagnoli MS RRT

BACKGROUND: The vital capacity (VC) has commonly been used as an indicator of ventilatory reserve in mechanically ventilated patients. However, because patient cooperation is required for a valid VC, the reliability of VC measurements is often questioned. We evaluated an alternate technique for the measurement of ventilatory reserve in intubated patients. METHODS: In consecutive ICU patients, we randomly compared ventilatory-reserve determinations by Method A: the standard VC (the maximal expired volume following maximal inspiration) and Method B: the volume inspired and expired immediately following a maximal inspiratory pressure (MIP) measurement. A Wright respirometer was used to measure volumes during both methods. Three measurements were made with Method A, and the largest was recorded. With Method B, we measured the inspired volume (IV) and expired volume (EV) on the first breath after a 20-second MIP measurement made with one-way valves. Sets of determinations were made at least 1 hour apart. Usable data were obtained on 64 patients, 30 women and 34 men, age 67 ± 13 years. RESULTS: Of the 64 patients, 39 were able to cooperate with Method A and 25 could not. In the cooperative group, the Method-A VC (982 ± 479 mL) was significantly greater than either the Method-B IV (841 ± 436 mL) ($p = 0.001$) or EV (900 ± 472 mL) ($p = 0.029$). However, in the uncooperative group, the Method-A VC (485 ± 332 mL) was significantly lower than the Method-B EV (610 ± 444 mL) ($p = 0.029$). In addition, for the total group, there was a moderate correlation (0.57) between the Method-A VC and the MIP, with VC predicted from the MIP by the equation: VC = (MIP) (17.6) + 84.2, although this accounted for only 32.5% of the variance in VC. CONCLUSIONS: In intubated patients unable to perform the VC maneuver, the ventilatory reserve determined by the expired volume after an MIP measurement was larger than the standard VC. In cooperative patients, the addition of 80 mL to the expired volume after an MIP measurement resulted in a volume equal to the VC. Furthermore, VC was moderately correlated to MIP; however, only about one third of the variance in VC was predicted by MIP alone. We do not recommend prediction of VC based solely on MIP. However, in the total group, each cm H_2O of MIP was equal to 20 mL of VC. (Respir Care 1991;36:1085-1092.)

Dr Kacmarek is Director, Respiratory Care Services, Massachusetts General Hospital; and Assistant Professor, Department of Anesthesia, Harvard Medical School—Boston, Massachusetts. Mr Foley and Ms Cheever are Staff Therapists; and Mr Romagnoli is Assistant Director, Administrative Services—Respiratory Care Services, Massachusetts General Hospital.

A version of this paper was presented by Mr Foley at the RESPIRATORY CARE OPEN FORUM during the 1990 AARC Annual Meeting in New Orleans, Louisiana.

Reprints: Robert M Kacmarek PhD RRT, Respiratory Care Services, Ellison 4, Massachusetts General Hospital, Boston MA 02119.

Background

Vital capacity (VC) is defined as the maximal volume expired after a maximal inspiration,[1] and provides an indication of an individual's ventilatory reserve.[2] In addition to the use of VC to characterize the severity of pulmonary disease,[3,4] many have suggested the use of VC to indicate a patient's ability to sustain spontaneous ventilation during recovery from acute ventilatory failure.[5-7] VC values below 10-15 mg/kg of body weight have been considered inadequate to sustain prolonged spontane-

ous ventilation,[5,6] although clinically we have seen patients with smaller VCs who were capable of such ventilation.

One of the major problems in the use of VC to evaluate ventilatory reserve is its dependence on patient cooperation and effort for accurate determination. In fact, in the pulmonary function laboratory, reproducibility is required for an acceptable test; according to American Thoracic Society standards, "the largest and second largest FVC from acceptable curves should not vary by more than 100 mL or 5% of the measurement, whichever is greatest."[8] In the ICU, it is difficult to ensure such reproducibility. Some of us have a cooperative patient perform a series of VC maneuvers, and then we report the largest value. In uncooperative patients, it is impossible to accurately evaluate the VC, although uncooperative patients may be precisely those in whom VC data are the most useful.

In the study reported here, we sought to determine (1) in cooperative patients, whether the inspired or expired volumes following a maximal inspiratory pressure (MIP) maneuver using a one-way valve system (allowing expiration but preventing inspiration) were equal to the forced VC, and (2) in uncooperative patients, whether the inspired or expired volume on the first breath after an MIP maneuver was larger than attempted VC measurements.

Methods

In a consecutive group of intubated patients in our medical ICU who were being weaned or ready to be weaned from mechanical ventilatory support, we compared ventilatory reserve determined by two methods. In Method A, patients were vigorously coached to inspire maximally, then to expire as completely and rapidly as possible. This maneuver was repeated three times, with the largest recorded VC used for the comparison. In Method B, we measured the inspired volume (IV) and expired volume (EV) during the first breath after a 20-second MIP measurement using a one-way valve system[9] that allowed expiration but prevented inspiration. The one-way valve system assures that the MIP measurement is made at the most optimal lung volume (at or below functional residual capacity [FRC]): this has been shown to result in greater MIP values than when a non-valved system is used.[9]

A random-numbers table was used to determine the order of method application.[10] Determinations for each method were performed at least 1 hour apart. All measurements were made by one of the four investigators (KF or PC). In order to ensure that data were available for early morning rounds (7:00 AM) and establishment of respiratory care plans, the initial set of data was obtained at the end of the night shift (6:00-7:00 AM). The second set was obtained approximately 1 hour later, on the day shift. Bias was eliminated by the performance of an equal number of Method-A and Method-B determinations on each shift and by each of the two investigators.

The setup for Method A is depicted in Figure 1A. An Airlife ventilator-monitoring adapter (consisting of a U-Adapit, a 22-mm-ID tube adapter, two one-way valves, and an oxygen-tubing connector) was attached to the patient's airway.* At the expiratory one-way valve, a 6-inch-long large-bore tube was affixed with a Wright respirometer to allow measurement of expired volume.

For Method B (Fig. 1B), we used a Boehringer manometer, oxygen-connecting tubing, an Airlife ventilator-monitoring adapter, two 6-inch-long large-bore tubes, and two Wright respirometers. To each end of the U-Adapit we attached a 6-inch-long large-bore tube, with a respirometer attached to the other end of each tube. The respirometers were configured in relation to the one-way valves to allow the measurement of IV and EV, as previously described by Greenhow.[11] During the MIP maneuver, the inlet port of the respirometer on the inspiratory limb was occluded. As the MIP determination continued, expiration occurred via the expiratory respirometer. After completion of the MIP measurement, occlusion of the inspiratory limb was released during the expiratory phase. The first unoccluded IV and the subsequent EV were recorded.

*Suppliers are identified in the Product Sources section at the end of the text.

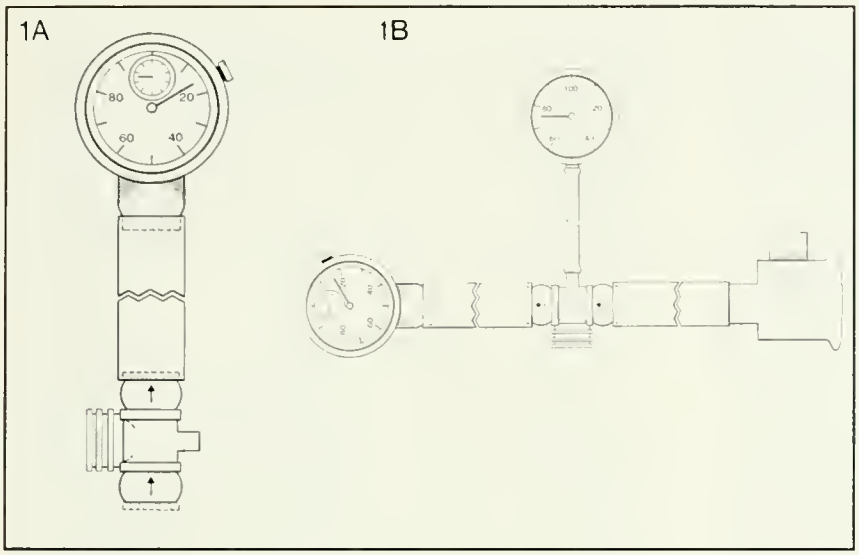

Fig. 1A. Setup for the determination of a standard VC in patients with artificial airways (Method A). 1B. Setup for the determination of MIP-induced inspiratory and expiratory volumes (Method B). Arrows indicate direction of flow through the U-Adapit.

The Boehringer manometers and Wright respirometers were those normally used in the ICU. Each received its normal preventive maintenance. No attempts were made to calibrate each instrument before each measurement; however, the same manometer and respirometer were used for all determinations on a given patient. We realized that these were not laboratory-standards instruments, but we used them because they reflected the level of accuracy noted clinically.

Experimental Protocol

A total of 76 patients was studied; however, 12 patients had to be dropped from the study because complete information was unobtainable as a result of their changing clinical status or the demands of the ICU. Thus, complete data were obtained on 64 patients with a mean age of 67 ± 13 years. Of these, 34 were men and 30 were women. Ten had COPD, 17 had cardiac disease, 10 were postoperative, and 27 had been admitted for other reasons (ie, neurologic disease, neuromuscular disease, gastrointestinal dysfunction, liver disease, and AIDS). There were no contraindications to the determinations of VC and MIP in any of the patients studied, and all measurements were performed as part of their routine care. Methods A and B were randomly used on each patient, with measurements made at least 1 hour apart. After measurements, patients were either mechanically ventilated or manually ventilated to ensure a rapid return to the premeasurement state. In addition, each patient was monitored for pulse and electrocardiogram, via a cardiac monitor, throughout each measurement period. In those patients in whom desaturation during stress had been documented, oxyhemoglobin saturation was monitored with a pulse oximeter. MIP or VC maneuvers were discontinued if frequent premature ventricular contractions, tachycardia, bradycardia, or arterial desaturation was noted. Patients were considered uncooperative if they were unable to follow commands given by the therapist performing the determination.

Statistical Analysis

All values for vital capacity were recorded in milliliters (mL), and means and standard deviations were determined for the cooperative and uncooperative subgroups. The significance of differences between methods was determined by the Student's *t* test,[10] with significance set at $p \leq 0.05$. Pearson product-moment correlations between MIP and VC, MIP and IV, and MIP and EV were determined for the total group, for the cooperative group, and for the uncooperative group.[10] Data analysis was performed with commercial software.

Results

The individual datapoints for each of the 64 subjects by each method are listed in Tables 1 and 2.

Of the 39 patients considered to be cooperative (Table 1), 21 were men and 18 were women; the group age was 66 ± 13 years. Of the 25 patients considered to be uncooperative (Table 2), 13 were men and 12 were women; the group age was 69 ± 13 years.

Table 3 shows that in the cooperative group, the VC (982 ± 479 mL) was significantly greater than the IV (841 ± 436 mL) ($p = 0.001$) or the EV (900 ± 472 mL) ($p = 0.029$). In the uncooperative group, the EV (610 ± 444 mL) was significantly greater than the VC (485 ± 332 mL) ($p = 0.029$).

The Pearson product-moment correlations between MIP and VC, MIP and IV, and MIP and EV for the total group ranged from 0.42 to 0.57, with the greatest (0.57) being for the relationship between MIP and VC (Table 4). However, the predictions of VC, IV, or EV from the MIP accounted for only 17.6 to 32.5% of the variance in VC, IV, or EV.

Discussion

The most important findings of this study were that (1) in the cooperative group, the first expired volume (900 ± 472 mL) following a MIP measurement, although significantly different from the VC determined by standard methods (982 ± 479 mL) ($p = 0.029$), differed from the VC (was lower) by only 80 mL; and (2) in the uncooperative group, the expired volume (610 ± 444 mL) significantly exceeded the VC determined by standard methods (485 ± 332 mL) ($p = 0.029$).

Because MIP was determined with a one-way valve system that allowed expiration but not inspiration, lung volume was reduced below FRC during the measurement, as noted by Marini et al.[12] This, in addition to the 20-second period of MIP measurement, produced sufficient stress to result in the equivalent of a vital capacity maneuver during the first breath after completion of the MIP measurement. The volume of gas inspired on the first breath after the MIP measurement was 60 mL less than the first exhaled volume for both subgroups. The reason for this difference between IV and EV cannot be determined from our data. It may be a result of stimulation of pulmonary stretch-receptors during the isometric contractions that occurred during the MIP maneuver. Receptor stimulation and the perception of suffocation may have enhanced expiration and rapid post-MIP breathing. In addition, it may be speculated that the MIP maneuver decreased lung volume not to residual volume (RV), but to somewhere between RV and the FRC level. However, regardless of the explanation, the EV appears to be a better estimate of VC than is the IV.

For both subgroups, three attempts at a standard VC were made, with vigorous coaching. This resulted in a higher VC in the cooperative group than in the uncooperative group. Based on our observations, the difference between VC and EV in the cooperative group appears to be the result of a more prolonged expiratory phase during the standard VC determination than during the MIP-induced determination. In the MIP-induced EV, expiration may have been more rapid as a result of ventilatory stress induced by the 20-second MIP maneuver; whereas, during the VC determination, coaching produced a slower, longer expiratory phase. Prior to the VC maneuver, the patient had not been stressed. In addition, the MIP-induced EV may have been lower than the VC in the cooperative group because of the flow resistance of the one-way valves. Greater resistance to flow would be encountered during a fast expiration (MIP method), resulting in a smaller volume's being expired. Thus, in this subgroup, the differences noted may be a result of the comparison of a fast to a slow VC, with the reduced volume measured by the MIP method simply a result of expiration's being prematurely ended by air hunger and system flow resistance. In the uncooperative group, the standard VC was lower than the MIP-induced EV because those patients could not properly perform the VC maneuver.

We found no previous data with which to compare our findings. However, the technique of evaluating EV after an MIP measurement was first described by Greenhow[11] in 1976, although that report did not include data comparing the IV or EV to VC.

Some authors[2,13] have proposed a relationship between VC and MIP. They indicate that an MIP of 20-25 cm H_2O is equivalent to a VC of 10-15 mL/kg of ideal body weight. Our data indicate a sig-

Table 1. Vital Capacity (VC), Inspired Volume and Expired Volume (IV and EV), and Maximal Inspiratory Pressure (MIP) in 39 Cooperative Patients*

		Method A	Method B		
Subject No./ Age/Sex	Diagnosis	VC (mL)	IV (mL)	EV (mL)	MIP (cm H_2O)
4/65/M	Cardiac	1000	800	1400	40
5/38/F	Other†	800	325	600	40
7/85/F	Cardiac	250	50	150	25
11/61/M	Surgical	1100	1000	1200	35
12/56/F	Surgical	900	700	850	20
13/57/M	Surgical	2000	1800	2100	60
14/73/M	Surgical	900	750	900	20
15/59/F	Surgical	1200	1000	1250	40
16/71/F	Surgical	600	600	525	40
17/81/M	COPD	500	400	500	42
18/62/F	COPD	720	800	400	25
20/68/F	COPD	1000	880	500	30
23/58/M	Cardiac	2000	2000	2000	60
24/44/F	COPD	1700	1700	1500	60
25/71/M	COPD	1000	1100	1000	60
28/66/M	Other	1600	600	800	52
30/83/M	Other	900	700	700	50
31/33/M	Other	1200	800	1200	50
32/74/M	Other	800	800	800	65
33/76/F	Cardiac	800	700	800	40
35/71/F	Other	1000	650	1000	60
36/44/M	Surgical	800	800	1000	55
37/74/M	Other	350	400	400	32
40/88/F	Other	750	800	700	50
41/58/F	Other	1000	1000	1100	47
42/61/M	COPD	700	700	700	60
43/86/M	Other	700	750	600	33
44/62/M	Cardiac	1900	1200	1200	60
45/51/F	Other	400	500	450	44
47/86/M	COPD	350	400	350	45
48/59/M	Other	1500	900	1500	43
49/66/F	Other	600	600	600	32
50/60/F	Other	1200	1200	1200	60
51/64/F	Cardiac	800	800	800	60
52/60/M	Cardiac	2000	2000	2000	60
60/77/F	Cardiac	400	350	340	35
61/65/M	Other	600	500	510	30
63/77/F	Other	800	550	500	40
Mean (SD) Age 66 (13)		980 (479)	841 (436)	900 (472)	45 (13)

* Cooperative = able to perform satisfactory vital capacity maneuver. IV = inspired volume after MIP measurement; EV = expired volume after MIP measurement.

† Other = neurologic or neuromuscular disease, gastrointestinal dysfunction, liver disease, or AIDS.

Table 2. Vital Capacity (VC), Inspired Volume and Expired Volume (IV and EV), and Maximal Inspiratory Pressure (MIP) in 25 Uncooperative Patients*

Subject No./ Age/Sex	Diagnosis	Method A	Method B		
		VC (mL)	IV (mL)	EV (mL)	MIP (cm H_2O)
1/86/M	COPD	400	400	600	10
3/88/M	Other†	350	300	400	20
6/88/F	Surgical	100	300	250	10
8/60/F	COPD	700	200	550	20
9/61/F	Other	150	300	320	20
10/44/F	COPD	600	300	900	40
19/69/F	Surgical	500	400	600	25
21/42/F	Other	600	500	1400	60
22/86/F	Cardiac	100	250	200	22
26/64/M	Other	1200	800	700	50
27/73/M	Other	300	350	400	60
29/71/M	Cardiac	1200	1900	2000	44
34/67/M	Cardiac	800	600	800	70
38/74/M	Cardiac	850	1200	1100	58
39/60/F	Cardiac	25	50	50	11
46/71/M	Other	350	1100	600	42
53/75/M	Surgical	750	800	1200	18
54/56/M	Cardiac	350	350	450	30
55/58/M	Cardiac	900	1100	800	42
56/68/F	Other	675	500	500	22
57/69/F	Cardiac	325	400	500	50
58/81/M	Other	150	200	200	25
59/62/F	Other	150	250	200	20
62/73/M	Cardiac	200	500	150	35
64/86/F	Other	400	700	400	18
Mean (SD) Age 69 (13)		485 (332)	550 (412)	610 (444)	33 (18)

* Uncooperative = unable to perform satisfactory vital capacity maneuver. IV = inspired volume after MIP measurement. EV = expired volume after MIP measurement.

† Other = neurologic or neuromuscular disease, gastrointestinal dysfunction, liver disease, or AIDS.

nificant but moderate correlation between VC and MIP for our total group of patients that, at best, predicts only 32.5% of the variance between MIP and VC. Other factors, most probably lung compliance and airways resistance, would have to be taken into account to improve prediction of VC. However, if we predict VC by using the equation for the total group [VC = (MIP) (17.6) + 84.2] for MIPs of 20 and 25 cm H_2O, VCs of 434 and 533 mL, respectively, are obtained. Assuming that the average weight of our ICU patients is 70 kg, and using the 10- to 15-kg relationship, MIPs of 20-25 cm H_2O would have produced VCs of 700-1,050 mL. Based on the large discrepancy between predicted and actual VC, we question the validity of this proposed relationship between MIP and VC and recommend that it not be used until further data are acquired.

Table 5 shows the results of dividing the VC and the EV by the MIP. Note that for the cooperative group, these calculations resulted in a value of

Table 3. Comparisons of Method A (VC) and Method B (MIP, IV, EV)*

	Cooperative Group† (n = 39)	Uncooperative Group (n = 25)
Age (y)	66 (13)	69 (13)
No. Sex	21 M, 18 F	13 M, 12 F
Method A		
VC (mL)	982 (479)	485 (332)
Method B		
MIP (cm H_2O)	45 (13)	33 (18)
IV (mL)	841 (436)‡	550 (412)
EV (mL)	900 (472)§	610 (444)‖
Diagnoses (No.)		
COPD	7	3
Cardiac	8	9
Surgical	7	3
Other¶	17	10

* VC = vital capacity; MIP = maximal inspiratory pressure; IV = inspired volume after MIP measurement; EV = expired volume after MIP measurement.

† Cooperative = able to perform satisfactory vital capacity maneuver. Uncooperative = unable to perform satisfactory vital capacity maneuver. All values are mean (SD) except sex and diagnoses.

‡ $p = 0.001$ VC vs IV (Student's *t* test).

§ $p = 0.029$ VC vs EV (Student's *t* test).

‖ $p = 0.029$ VC vs EV (Student's *t* test).

¶ Other = neurologic or neuromuscular disease, gastro-intestinal dysfunction, liver disease, or AIDS.

about 20 mL/cm H_2O; whereas, in the uncooperative group, the value was markedly reduced—to 14.7 mL/cm H_2O for VC/MIP and 18.5 mL/cm H_2O for EV/MIP. However, as Table 5 also shows, if 80 mL (the difference between the cooperative group's VC and EV) is added to the uncooperative group's EV, the resulting volumes divided by the MIP equals the VC divided by the MIP of the cooperative group: (EV + 80)/MIP = 20.9 mL/cm H_2O. These data lead us to propose the following in adult medical ICU patients: (1) in uncooperative patients, the EV determined after an MIP measurement better represents ventilatory reserve than does a standard VC; (2) in uncooperative patients, the addition of 80 mL to the MIP-induced EV results in a volume equivalent to the actual VC; and (3) each cm H_2O of MIP in cooperative as well as uncooperative patients is equal to about 20 mL of VC. Because effective static compliance and airways resistance were not determined for these patients, and because the group was so diversified (with small numbers in each subgroup), it is impossible to generalize the 20 mL/cm H_2O VC value to other groups. However, this value may be representative of the relationship between VC and MIP in similar groups of ICU patients ready to wean from ventilatory support, although additional data are required before these conclusions can be generalized.

Table 4. Correlations between MIP and VC, MIP and IV, and MIP and EV in 64 ICU Patients*

Group	Predictor	Coefficient	Constant	Resultant	Correlation	Significance	r^2
All patients	MIP	17.59	84.2	VC	0.57	$p < 0.001$	0.325
All patients	MIP	15.13	121.8	IV	0.54	$p < 0.001$	0.292
All patients	MIP	16.74	117.3	EV	0.55	$p < 0.001$	0.303
Cooperative patients†	MIP	18.92	138.3	VC	0.51	$p < 0.001$	0.260
Cooperative patients	MIP	18.06	35.5	IV	0.53	$p < 0.001$	0.281
Cooperative patients	MIP	19.54	28.9	EV	0.53	$p < 0.001$	0.281
Uncooperative patients	MIP	9.44	174.7	VC	0.50	$p < 0.010$	0.250
Uncooperative patients	MIP	9.92	223.8	IV	0.42	$p < 0.050$	0.176
Uncooperative patients	MIP	12.10	211.9	EV	0.48	$p < 0.050$	0.230

* MIP = maximal inspiratory pressure; VC = vital capacity; IV = inspired volume after MIP measurement; EV = expired volume after MIP measurement. VC = Method A; MIP, IV, EV = Method B.

† Cooperative = able to perform satisfactory vital capacity maneuver (n = 39). Uncooperative = unable to perform satisfactory vital capacity maneuver (n = 25).

Table 5. Relationship of VC and EV to MIP in 64 ICU Patients*

Group	VC/MIP	EV/MIP	(EV + 80)/MIP
Cooperative†	21.8	20.0	—
Uncooperative	14.7	18.5	20.9

* VC = vital capacity; MIP = maximal inspiratory pressure; EV = expired volume after MIP measurement; EV + 80 = MIP-induced expired volume + 80 cm H_2O. All values determined by the use of the means for each measurement.

† Cooperative = able to perform satisfactory vital capacity maneuver. Uncooperative = unable to perform satisfactory vital capacity maneuver.

Conclusions

In patients unable to cooperate in the standard vital capacity maneuver to permit estimation of ventilatory reserve, we found that the addition of 80 mL to the expired volume after a maximal inspiratory pressure maneuver resulted in a volume equal to the standard VC. A moderate correlation between MIP and VC for the total group was found. However, because only 32.5% of the variance in VC could be related to the MIP measurement, we do not recommend that VC be predicted solely on MIP measurement. However, in our patients who were able to cooperate in producing a valid vital capacity, each cm H_2O of MIP was equal to about 20 mL of vital capacity.

PRODUCT SOURCES

Ventilator Monitoring Adapter:
U-Adapit, Baxter Hospital Supply, Evanston IL

Large-Bore Tubing:
Professional Medical Products Inc, Greenwood SC

Oxygen Connecting Tubing:
Baxter Hospital Supply, Evanston IL

Manometer:
Boehringer Laboratories Inc, Norristown PA

Respirometer:
Wright, Ferraris Medical, Holland NY (formerly marketed by Fraser Harlake Inc, Orchard Park NY)

Statistical Software:
PC Statistician, Human Systems Dynamics, Northridge CA

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Effect of PEEP-Valve Placement on Function of a Home-Care Ventilator

Jon W Gietzen BS RRT, John A Lund BA RRT, and Jon L Swegarden BS RRT

BACKGROUND: The addition of a PEEP valve to the circuit of a home-care ventilator like the Aequitron LP-6 can be viewed as a consumer modification of the system. We sought to determine the effect that such a modification would have on ventilator function. METHODS & MATERIALS: Part 1. We tested the effect of PEEP level and PEEP-valve position on volume delivered at the ventilator Y-adapter, over a range of tidal volumes. Part 2. We held tidal volume, frequency, and inspiratory time constant, and varied PEEP level and PEEP-valve position to test the effect of PEEP-valve position on pressures measured at the ventilator outlet, patient-Y, and within the exhalation-valve pressurization line. RESULTS: Conventional placement of the PEEP valve (distal to, or 'after,' the exhalation valve) in the LP-6 ventilator circuit resulted in statistically significant and potentially clinically important decreases in the volumes delivered to the patient at some ventilator settings. Proximal placement of the PEEP valve (proximal to, or 'before,' the exhalation valve) resulted in consistent volumes delivered to the patient at all levels tested, without changing the ventilatory performance characteristics of the ventilator as reflected by pressure waveforms. CONCLUSION: We recommend that appropriate observations and measurements be made to verify system function before a home-care ventilator modified to provide PEEP is applied to the patient. (Respir Care 1991;36:1093-1098.)

Background

Assuring proper ventilator performance is an essential facet of the care of the long-term ventilator patient.[1] In recent years, so-called home-care ventilators (electrically powered, piston-driven, volume-limited[2]) have come into common use for ventilator-dependent patients both in institutions and in the home.[3] Commonly available home-care ventilators (such as the Aequitron LP-6; Lifecare PLV-100, PLV-102, and PVV; Puritan-Bennett Companion; and Thompson M25A and M25B) are not designed with the intrinsic capability to provide positive end-expiratory pressure (PEEP).[2] If the application of PEEP is desirable, perhaps to improve oxygenation or to help prevent atelectasis, a PEEP valve must be added to the system.[3]

We observed that when PEEP was applied in the conventional manner (PEEP valve distal to the exhalation valve) to an Aequitron LP-6 home-care ventilator,* which had passed a circuit leak test,[4] a discrepancy was seen between delivered tidal volume ($V_{T\text{-del}}$, the volume measured at the outlet of the ventilator) and 'lung' tidal volume ($V_{T\text{-lung}}$, the volume measured at the interface of the patient and the patient-Y that could be clinically important). We postulated that the reason for the discrepancy rested with the effect of the position of the PEEP-valve to seal completely during the inspiratory phase of a mechanical breath.

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*Suppliers are identified in the Product Sources section at the end of the text.

Methods and Materials

Description of Devices

We attached a Baxter two-limb disposable circuit to the Aequitron LP-6 ventilator. No humidifier was included. Tubing compliance measured approximately 1.5 mL/cm H_2O [0.15 L/kPa].[4] After the circuit had been shown to be leak free,[4] a Manley test lung was attached, with compliance set at 50 mL/cm H_2O [0.50 L/kPa] and resistance at 0 cm $H_2O \cdot s \cdot L^{-1}$ [0 kPa · s · L^{-1}]. We performed the evaluation in two parts. Three exhalation valves (Figs. 1-3)—a Puritan-Bennett permanent, a Baxter disposable, and a Seamless-Dart disposable exhalation valve—were tested on the exhalation limb of the circuit. We compared the effect on $V_{T\text{-lung}}$ of distal PEEP-valve (Fig. 1) placement to the effect of proximal PEEP-valve placement (Fig. 4). PEEP was applied to the circuit using a Vital Signs PEEP valve. We chose the LP-6 because it (like other home-care ventilators such as Lifecare's PLV-100, PLV-102, and PVV; Puritan-Bennett Companion; and Thompson M25A and M25B) pressurizes the exhalation valve during inhalation at the same pressure supplied prior to the ventilator gas outlet (Fig. 5).[2]

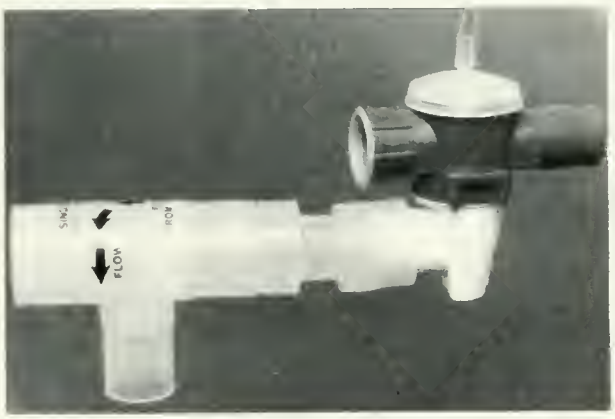

Fig. 1. Seamless-Dart disposable exhalation valve with PEEP valve in distal position.

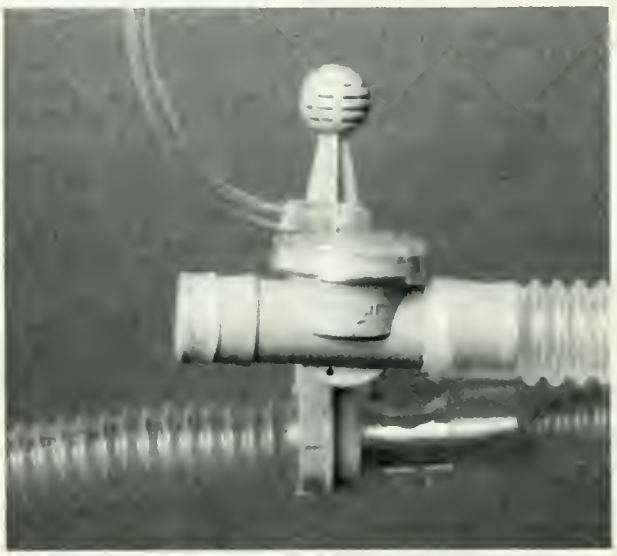

Fig. 2. Baxter disposable exhalation valve.

Fig. 3. Puritan-Bennett exhalation valve.

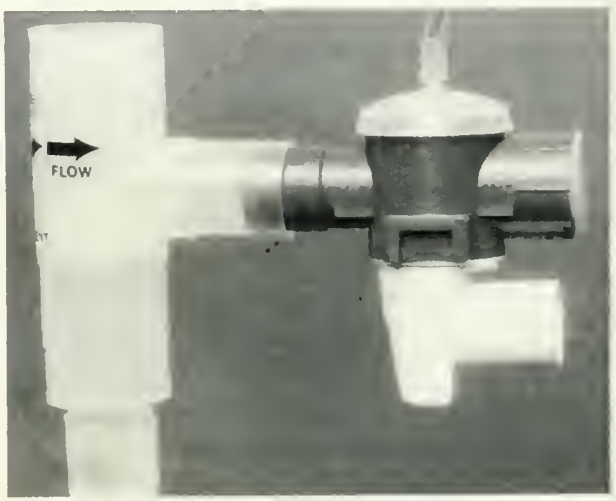

Fig. 4. Seamless-Dart disposable exhalation valve with PEEP valve in proximal position.

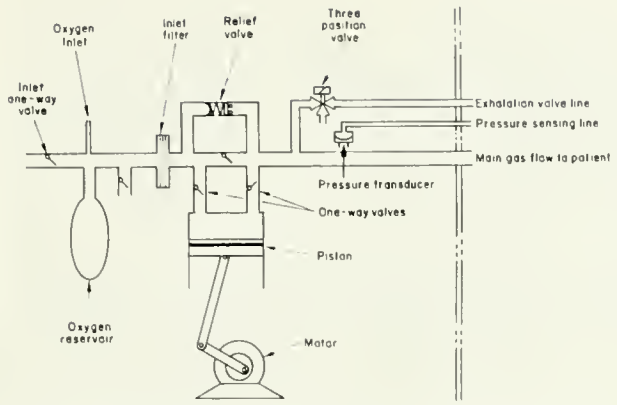

Fig. 5. Functional diagram of the LP-6 ventilator. (Used with the permission of Aequitron Medical Inc.)

Evaluation Methods

Part 1. We documented the effect of PEEP-valve position and PEEP level on $V_{T\text{-lung}}$, over a range of set tidal volumes ($V_{T\text{-set}}$). Ventilator mode (assist/control), frequency (f, 12 breaths/min), and inspiratory time (t_i, 0.8 s) were constant. We varied the $V_{T\text{-set}}$ (from 200 to 800 mL), the level of PEEP (from 2.5 to 5.0 cm H_2O or [0.25 to 0.50 kPa]), and the position of the PEEP valve. Once the ventilator had been adjusted to a given tidal volume and had delivered at least 5 breaths, the $V_{T\text{-del}}$ was measured. To measure $V_{T\text{-del}}$, we placed a calibrated 100 L/min pneumotachograph attached to a Lung Mechanics Monitor at the outlet of the ventilator. To measure $V_{T\text{-lung}}$, the pneumotachograph was then placed at the point of interface of the patient-Y and the test lung. Peak inspiratory pressure (PIP) and PEEP were measured simultaneously using the lung mechanics monitor. Four measurements were recorded for each setting.

We also measured $V_{T\text{-lung}}$ with a commonly available vane-type respirometer to establish the correlation with the measurements made by the 100 L/min pneumotachograph.

Statistical Analysis

A comparison was made of the mean and standard deviation of the $V_{T\text{-lung}}$ and PIP comparing distal PEEP-valve to proximal PEEP-valve values at each set tidal volume, using a 2-tailed Student's *t* test with 8 degrees of freedom. The volumes measured by the vane spirometer and the pneumotachograph were compared. A test for linearity was performed using a linear correlation regression formula with Givens transformations using Linpack routines. The test for linearity had 11 degrees of freedom.

Part 2. Frequency (f, 12 breaths/min), inspiratory time (t_i, 0.8 s), PEEP (5.0 cm H_2O [0.50 kPa]), mode (assist/control), and tidal volume delivered ($V_{T\text{-del}}$) were held constant. PEEP-valve position was varied between the proximal and distal positions. Simultaneous pressure recordings were made throughout the test to document any changes occurring in the pressure-time waveforms. The pressure recorder was set on the 100-torr scale at a speed of 25 mm/s. Three pressure transducers were calibrated to atmosphere with the pressure-transducer diaphragm level with each site monitored. The transducers were placed at (1) the gas outlet of the ventilator, (2) the interface of the Y-adapter and the test lung, and (3) the exhalation-valve pressurization line (Fig. 6).

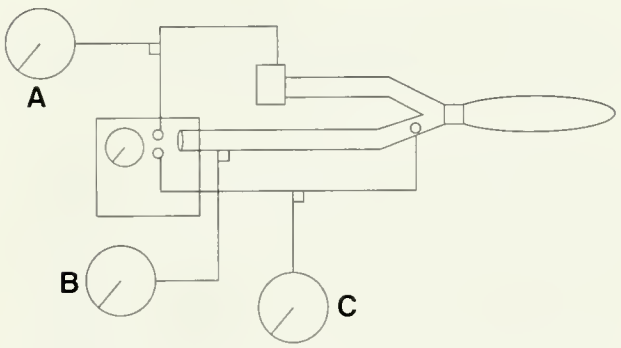

Fig. 6. Sites at which pressures were measured during Part 2 of the study. A. The pressure transducer was teed into the exhalation line. B. The pressure transducer was teed into the gas outlet of the ventilator. C. The pressure transducer was teed into the interface of the Y-adapter with the test lung.

Results

The effects of PEEP-valve position are seen in Table 1. The position of the valve did not significantly affect the volumes delivered by the Bennett valve ($p < 0.01$). The Seamless-Dart valve was significantly affected by the position of the PEEP

Table 1. Comparisons of the Effects of Two PEEP-Valve Positions and Two Levels of PEEP on Preset, Delivered, and Received Tidal Volumes with a Home-Care Ventilator

	0 PEEP			Proximal 2.5 cm H_2O		Distal 2.5 cm H_2O		Distal 5.0 cm H_2O		Proximal 5.0 cm H_2O	
Set V_T	$V_{T\text{-del}}$	$V_{T\text{-lung}}$	PIP	$V_{T\text{-lung}}$	PIP	$V_{T\text{-lung}}$	PIP	$V_{T\text{-lung}}$	PIP	$V_{T\text{-lung}}$	PIP
Puritan-Bennett											
200	160 (5.5)	153 (5.2)	4.5 (0.2)	157 (5.9)	4.6 (0.2)	154 (6.0)	4.5 (0.2)	145 (5.2)	7.3 (0.2)	149 (5.2)	8.8 (0.2)
450	379 (3.3)	367 (5.9)	12.9 (0.2)	367 (5.2)	13.0 (0.2)	362 (4.5)	12.9 (0.1)	357 (5.2)	18.2 (0.4)	353 (5.2)	20.1 (.3)
600	574 (6.7)	549 (3.3)	23.0 (0.3)	548 (6.3)	23.2 (0.4)	543 (1.7)	23.2 (0.0)	530 (2.0)	31.2 (0.3)	522 (4.5)	32.4 (0.3)
800	783 (6.0)	735 (3.3)	38.0 (0.4)	732 (2.8)	38.2 (0.4)	727 (1.7)	37.6 (0.0)	710 (2.0)	51.0 (0.4)	704 (2.8)	49.8 (0.4)
Seamless											
200	160 (5.5)	153 (5.1)	4.3 (0.2)	156 (5.8)	4.4 (0.2)	163 (4.5)	4.7 (0.1)	92.8 (3.2)	5.6 (0.1)	155 (5.0)	10.4 (0.2)
450	379 (3.3)	366 (6.0)	12.6 (0.2)	369 (5.2)	12.9 (0.1)	365 (5.2)	12.7 (0.1)	333 (5.2)	15.7 (0.3)	358 (4.5)	19.5 (0.3)
600	574 (6.7)	539 (1.7)	23.1 (0.2)	541 (3.3)	23.2 (0.3)	540 (2.8)	23.1 (0.2)	484 (0.0)	29.0 (0.2)	528 (2.8)	32.6 (0.4)
800	783 (6.0)	736 (4.0)	38.0 (0.4)	735 (3.3)	38.4 (0.0)	737 (3.3)	38.4 (0.0)	688 (2.8)	43.6 (0.4)	713 (1.7)	53.6 (0.6)
Baxter											
200	160 (5.5)	137 (6.0)	4.1 (0.2)	117 (6.0)	3.5 (0.2)	144 (5.3)	4.3 (0.2)	84.8 (2.3)	6.4 (0.0)	153 (3.7)	10.1 (0.2)
450	379 (3.3)	368 (4.9)	12.9 (0.2)	366 (6.0)	12.9 (0.2)	366 (4.5)	13.2 (0.2)	360 (4.9)	19.7 (0.3)	361 (7.1)	19.2 (0.3)
600	574 (6.7)	546 (2.0)	23.4 (0.2)	546 (3.5)	23.7 (0.3)	542 (2.0)	23.7 (0.2)	531 (4.4)	32.3 (0.2)	528 (2.8)	34.2 (0.4)
800	783 (6.0)	741 (3.3)	38.6 (0.4)	739 (1.7)	38.8 (0.4)	733 (3.3)	39.0 (0.4)	722 (4.5)	48.6 (0.4)	714 (3.5)	47.2 (0.6)

* All values are mean (SD). $V_{T\text{-del}}$ = volume measured at ventilator outlet. $V_{T\text{-lung}}$ = volume measured at "patient"-Y in mL (ie, V_T received). PIP = peak inspiratory pressure in cm H_2O.

through all tidal volumes levels tested when 5.0 cm H_2O of PEEP was applied ($p < 0.01$). The Baxter valve was affected significantly at the 200-mL tidal volume setting ($p < 0.01$). When the PEEP valve was positioned in the proximal position to the exhalation valve the $V_{T\text{-del}}$ and $V_{T\text{-lung}}$ were comparable with a 99.8% correlation. The PIP did not vary significantly with the Bennett valve ($p < 0.01$). The PIP generated with use of the Seamless-Dart valve was significantly different over all tidal volumes tested when 5.0 cm H_2O [0.5 kPa] of PEEP was applied. The PIP varied with the Baxter valve when the tidal volume was set at 200 mL ($p < 0.01$).

As can be seen in Figure 7, volumes measured with the Wright respirometer compared closely to values obtained with the pneumotachograph, demonstrating a correlation of 99.7%.

In Part 2 of the study, we were able to document a difference in the shape of the pressure waveforms generated dependent upon PEEP-valve placement. The pressure waveforms generated without PEEP were considered the baseline waveforms. When the circuit was operated with PEEP applied distally, the pressure tracings reveal that peak pressure in

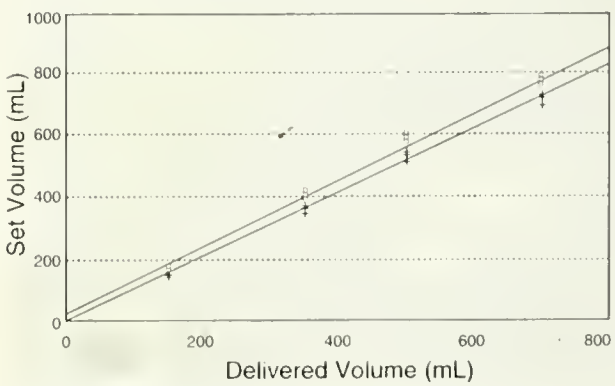

Fig. 7. Comparison of delivered volumes as measured by pneumotachograph —+— and by vane spirometer —□—.

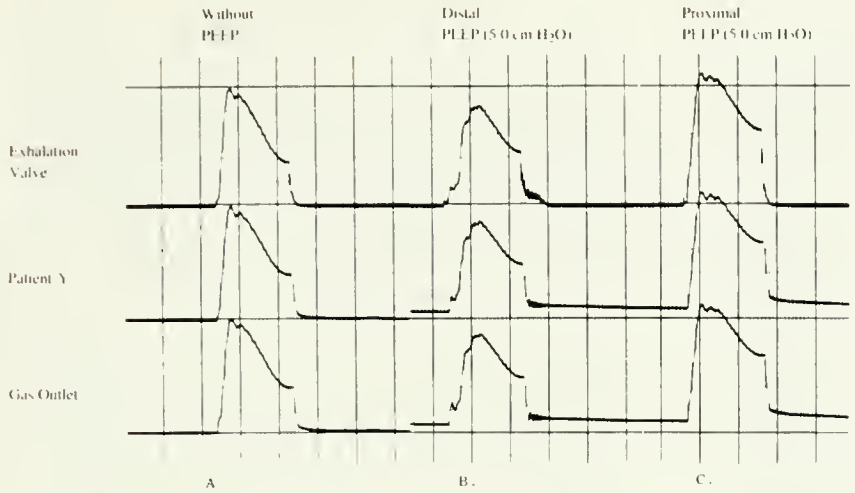

Fig. 8. Comparison of pressure waveforms generated using the Seamless-Dart disposable valve at the same settings comparing effects of A. 0 PEEP; B. PEEP of 5.0 cm H_2O [0.50 kPa], with PEEP valve distal; and C. PEEP of 5.0 cm H_2O [0.50 kPa], with PEEP valve proximal.

the patient circuit is reduced in spite of the elevated baseline due to the PEEP. When PEEP was applied in the proximal position (before the exhalation valve), the pressure-time traces resume the baseline (Fig. 8).

Discussion

The addition of a PEEP valve to the circuit of a home-care ventilator, like the Aequitron LP-6, can be viewed as a consumer modification of the system. Any modification such as this must be carefully scrutinized to determine whether the overall function of the system has been affected in any way. PEEP is commonly provided to the system by attaching a PEEP valve distal to the exhalation valve in a two-limb ventilator circuit. Distal placement of the PEEP valve in a ventilator in which the exhalation-valve line is pressurized by gas at the same pressure as that supplied to the ventilator gas outlet resulted in clinically important and statistically significant loss of $V_{T\text{-lung}}$. In the conditions in which the leak developed, it appeared that the exhalation-valve assembly did not seal completely.

This failure to seal completely can be explained by outlining the steps that the LP-6 ventilator uses to pressurize the exhalation-valve assembly during the inspiratory phase of a mechanical breath. Once the piston in the ventilator has reached the volume level set prior to the beginning of inspiration, it begins to compress the gas in the piston chamber, opening the one-way valve that leads to the gas outlet of the ventilator. This pressurized gas flow simultaneously to the patient circuit and to a valve that controls the flow of gas to the exhalation-valve line. Throughout the inspiratory phase as the pressure increases (as a consequence of piston motion and lung mechanics), the pressure in the patient side of the exhalation-valve diaphragm rises as pressure also rises in the ventilator side of the exhalation-valve dia-phragm. With both sides of the exhalation-valve diaphragm exerting equal and opposite pressures, the valve remains sealed and no gas flows out of the exhalation valve. When inspiration ends and expiration begins, the valve controlling pressure to the exhalation-valve line vents to the atmosphere, thus enabling the patient side of the exhalation valve to open and allow gas to be exhaled. This sequence of events repeats itself whenever the ventilator provides a mechanical breath.

However, when PEEP is applied to the system in the distal position, a pressure gradient develops—preventing the exhalation-valve diaphragm, under certain operating conditions, from sealing adequately. The reason is that the patient-circuit side of the diaphragm maintains the level of PEEP set, but the ventilator side of the exhalation-valve diaphragm continues to vent to atmospheric pressure. With the PEEP valve placed distally, the patient side of the diaphragm has an extra amount of pressure that must be overcome prior to proper sealing of the exhalation valve. The Bennett valve had no significant change in V_T when PEEP was applied distally or proximally; however, both the Baxter and the Seamless-Dart exhalation valves intermittently allowed significant loss in $V_{T\text{-lung}}$. Each valve appears to incorporate different design characteristics that possibly change performance.

Other possible sources of leaks or contributing causes must be ruled out when troubleshooting for this type of problem. These sources can be internal malfunctions of the ventilator (eg, piston stalling) or external from the ventilator (eg, leak in humidifier or tubing). Once the practitioner is satisfied that such sources do not contribute in any way to the problem, modifications in use (eg, open- or closed-IMV setups, continuous flow, pressure limit-relief valves) should be evaluated.[2-4]

Although vane respirometers may not have the accuracy and reproducibility of a pneumotachograph,[2] they should function reliably enough to provide a trendable measurement that enables the clinician to assess whether the addition of PEEP is adversely affecting the $V_{T\text{-lung}}$ and to make timely adjustments to fulfill patient requirements (Fig. 7).

By 'teeing' into the exhalation-valve pressure line with a pressure manometer, the practitioner can compare the pressures generated on the patient side of the valve (from the ventilator pressure manometer) and the ventilator side of the valve (Fig. 6). What should be observed are that equal pressures are exerted in both manometers simultaneously for each mechanical breath. Prior to the initiation of a mechanical breath, if the ventilator side of the exhalation valve does not approximately equal the pressure on the patient side of the exhalation valve, a leak may occur during inspiration. This may cause a clinically important portion of the tidal volume to pass through the exhalation valve during the mechanical breath.

Conclusion

Any modification of equipment designed for human use deserves a thorough evaluation for potential complications from the modification. Our study shows that distal placement of the PEEP valve in the LP-6 ventilator circuit, under certain conditions, decreases the tidal volume made available to the patient. We suggest proximal placement of the PEEP valve (before the exhalation valve) for PEEP application with home-care ventilators. Placement of the PEEP valve in this position resulted in proper function of the ventilator system throughout all settings tested. Verification of proper function should be undertaken before any home-care ventilator is applied to a patient.

ACKNOWLEDGMENTS

We thank Gary Lee RRT, Gary Brown RRT, Kevin Dahlgren RN, Dave Gausman RN, Mike Leier RRT, GF Atwood MD, Wm C Rosen MD, BL March MD of St Luke's Hospital-MeritCare, The Children's Hospital-MeritCare Advisory Board, and S Zhang PhD of Moorhead State University, Moorhead, Minnesota.

PRODUCT SOURCES

Ventilator:
Aequitron LP-6, Aequitron Medical Inc, Minneapolis MN

Lung Model:
Manley Test Lung, Medishield, Harlow, Sussex, England

Exhalation Valves:
Puritan-Bennett permanent, Puritan-Bennett Corp, Overland Park KS
Baxter disposable, Baxter Healthcare Corp, Valencia CA
Seamless-Dart disposable, Seamless-Dart, Div of Professional Medical Products, Ocala FL

PEEP Valve:
Vital Signs Inc, Totowa NJ

Transducers:
Spectromed disposable, Spectromed Inc, Oxnard CA

Monitor and Recorder:
HP 8890B Cath Lab System, Hewlett-Packard, Waltham MA
HP 7760A Recorder, Hewlett-Packard, Waltham MA

Flow and Volume Measuring Devices:
Wright respirometer, Ferraris Medical, Holland NY
Pneumotachograph, 700 series 0-100 L/min, Hans Rudolph Inc, Kansas City MO
2600 Infant Lung Mechanics Monitor, SensorMedics, Yorba Linda CA

Ventilator Circuit:
Baxter Isothermal Custom Adult 1372-H08, Baxter Healthcare Corp, Pharmaseal Div, Valencia CA

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Cost Impact of Metered Dose Inhalers vs Small Volume Nebulizers in Hospitalized Patients: The Cleveland Clinic Experience

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To assess the cost impact of using metered dose inhalers (MDIs) versus small volume nebulizers (SVNs) for hospitalized adult patients not being managed in ICUs, we analyzed the labor, equipment, and medication costs associated with using MDIs at The Cleveland Clinic Foundation. Over the study interval (January 1988-December 1989), a policy was implemented to enhance MDI use, resulting in increased use of MDIs (18% of all bronchodilator treatments in 1989 vs 5% in 1988). Based on a volume of approximately 70,000 bronchodilator treatments/year in our hospital, increased MDI use with this policy reduced direct costs by $26,510, with associated savings in respiratory-therapist time. To extend this analysis of costs to other institutional settings, we present an analysis of projected changes in institutional costs when the volume of bronchodilator therapies and the percentage administered by MDI varies. (Respir Care 1991;36:1099-1104.)

Introduction

Despite preferential use of nebulized bronchodilators in our institution and others,[1] recent literature suggests that adequate delivery of bronchodilators by metered dose inhaler (MDI) can provide equal efficacy in a variety of clinical settings, including the treatment of patients with acute exacerbations of asthma or chronic obstructive pulmonary disease,[2-6] and patients with stable chronic obstructive pulmonary disease.[1,7,8] Previous analyses of institutional costs in converting from small volume nebulizer (SVN) to metered dose inhaler in patients not receiving care in the intensive care unit (ICU) suggests considerable savings associated with MDI bronchodilator use.[2,4,8] Specifically, at Cedars-Sinai Medical Center, Jasper et al[4] estimated that substitution of MDI therapy for nebulized bronchodilator therapy would result in a net direct cost savings of $253,487 per year, based on 1985 utilization data. More modest but definite institutional cost savings for equally effective bronchodilation is also suggested by two other, less extensive analyses.[2,8]

Our research examines the institutional direct costs incurred by the MDI policy currently (1991) employed at The Cleveland Clinic Foundation. To extend prior cost audits,[2,4] our analysis examines the 2-year trends in costs associated with bronchodilator therapy after a policy to promote MDI use had been implemented. Estimates of labor costs use real-time measurements based on a hand-held computer system (CliniVision System, Puritan-Bennett, Carlsbad CA). Finally, to make this analysis more generalizable to hospitals of different siz-

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es and with different MDI policies, the current analysis considers the cost impact of MDI use over a spectrum of hospital settings.

Methods

Our analysis was limited to data from MDI and SVN use by the Respiratory Therapy Section at The Cleveland Clinic Foundation from January 1, 1988, through December 31, 1989. Because the majority (94%) of inpatient aerosolized bronchodilator prescriptions were for β_2 agonists and anticholinergic medications (ie, ipratropium bromide [Atrovent]), the analysis of MDI and SVN use was confined to these two types of drugs.

Under the guidelines of the Respiratory Therapy Section, therapists help assign the optimal method of delivering inhaled medications when a specific delivery method has not been specified by the ordering physicians. As a general rule, MDIs are prescribed whenever possible (ie, when the necessary medication is available by MDI, and the patient is an appropriate candidate to use an MDI effectively).[9-17] To enhance MDI use, all MDIs are administered with a spacer device (Inspir-Ease, Schering Corp, Kenilworth NJ).[9,10,12,13,16,17] Patients deemed unable to use the MDI-spacer-system effectively received prescribed medication through an SVN.[18,19] SVNs are changed routinely every 3 days or sooner if needed (eg, when medication refluxes into tubing). Albuterol sulfate, the bronchodilator most frequently administered in SVNs, was provided in unit doses (2.5 mg of albuterol sulfate prediluted in normal saline to a total volume of 3 mL, or 0.83 mg/mL).

Once the initial drug delivery mode has been prescribed, the respiratory therapist instructs the patient in the appropriate technique and then assesses the patient's technique for self-administration. When an MDI is prescribed but the patient's performance reveals that he cannot use the MDI adequately, an SVN is substituted after the therapist communicates with the ordering physician.

The costs cited in the current analysis reflect direct institutional expenses incurred in providing bronchodilator treatments, by either MDI or SVN. Issues of revenue recapture and external reimbursement for services are deemed outside the scope of the analysis.

In the analysis of labor costs, distinctions are made between the therapist's initial instructional time (when detailed instructions on drug delivery techniques are given, the patient is assessed, and the patient demonstrates his or her ability to use proper technique) and the subsequent treatment and remedial instruction visits (when the therapist monitors the patient's self-administration of each ordered dose). Work-load excesses precluded only 2% of treatment visits, and missed treatment visits by the therapist are excluded from the calculations below.

Data regarding the volume of treatments and therapist time to administer treatments were tracked by the Respiratory Therapy Section's computer system that incorporates hand-held terminals.

Results

Costs of Medication, Drug Delivery, Equipment, and Labor

Table 1 presents the average direct costs for medication, delivery system equipment, and therapist labor for the period January 1988 through December 1989. Based on an average salary of $13.75/hour (excluding benefits) for a registered respiratory therapist, the labor costs were $0.229/min.

The mean time for initial instruction with SVN and MDI was the same (10 min, range 7-22 min). The times for treatment and remedial instruction were longer with SVN (mean 13 min, range 9-25 min) than with MDI (mean 5 min, range 3-12 min), largely reflecting the longer time necessary to administer medications with SVN than with MDI.

Medication costs (Table 1) are based on our strategy of using single MDI canisters in multiple patients (ie, the patient inserts the canister into his or her own InspirEase spacer device). The cost/dose (two puffs $0.035) of an MDI is based on the most commonly used inhalers (ie, metaproterenol, salbutamol, and ipratropium bromide), and the cost of a unit-dose bronchodilator with SVN ($0.49/treatment) is based on the cost of a unit dose of albuterol sulfate in our institution.

Table 1. Cost Estimates for Metered Dose Inhaler (MDI) and Small Volume Nebulizer (SVN) Use

	MDI	SVN
Labor		
Average hourly rate for RRT (without benefits)	$13.75	$13.75
Initial patient instruction	2.29*	2.29*
Subsequent treatment & remedial instruction	1.15†	2.98‡
Medication		
Unit-dose (per treatment)	NA	0.49
MDI (2 puffs/treatment)	0.035	NA
Equipment		
SVN kit (each)	NA	1.10
Use (per treatment)		0.09
Storage bag (each)	0.12	0.12
Use (per treatment)	0.01	0.01
Spacer device (each)	3.16	NA
Use (per treatment)	0.26	NA
Total labor, medication, & equipment cost/treatment		
Initial instruction + treatment	3.75	5.86
Subsequent treatment & remedial instruction (each)	1.46	3.57

*Mean time 10 min, range 7-22 min.
†Mean time 5 min, range 3-12 min.
‡Mean time 13 min, range 9-25 min.

Equipment costs (Table 1) associated with MDI use reflect cost of the spacer and the bag in which the setup is stored. For SVN users, the equipment cost reflects the SVN and storage bag. Total cost for 1989 spacers (without storage bag) was $3,309, in contrast to $5,199 expended on SVN equipment. This greater cost/treatment for the MDI ($0.26/treatment vs $0.09/treatment for SVN) somewhat offsets the substantial savings associated with MDI use based on labor and medication costs. Despite this offsetting equipment expense, the institutional cost per patient (an average of 12 treatments/patient, 4 times daily for 3 days) for MDI use ($17.52) was less than for SVN ($42.84). Overall, costs/initial instruction and evaluation for MDI use were $3.75 vs $5.86 for SVN instruction and evaluation and $1.46 vs $3.57, respectively, for subsequent treatment and remedial instruction visits.

The total volume and direct costs associated with the use of MDI and SVN by our Section during 1988 and 1989 are itemized in Table 2. Over this interval, the proportion of total therapies administered by MDI rose from 5 to 18%, reflecting a reinforced policy to encourage MDI use when deemed appropriate.

During 1989, a total of 56,712 follow-up SVN treatments were administered utilizing 12,288 person-hours with an associated labor cost of

Table 2. Cost Comparisons for Bronchodilator Therapy by Metered Dose Inhaler (MDI) and Small Volume Nebulizer (SVN) at The Cleveland Clinic Foundation during 1988-1989

	MDI	SVN	Total
Treatment Data			
Number of patients, 1988	283	5,310	5,593
Number of patients, 1989	1,047	4,726	5,773
Number of treatments, 1988	3,396	63,725	67,121
Number of treatments, 1989	12,564	56,712	69,276
Cost of treatments, 1988*	$5,606	$239,658	$245,264
Cost of treatments, 1989*	$20,741	$213,284	$234,025
Total bronchodilators (%), 1988	5	95	100
Total bronchodilators (%), 1989	18	82	100
Cost & Time Savings			
Cost savings from providing MDI therapy†			
1988	$7,166		
1989	$26,510		
Reduction in cost (%) 1989 over 1988		4.58%	
Person-hours saved by providing MDIs			
1988	453		
1989	1,675		

*Includes labor costs for initial and subsequent treatments, medications, and equipment.
†(Cost of MDI treatments at SVN cost) – (number of MDI treatments)(savings/MDI treatment) = cost savings.

$169,002. By comparison, a total of 12,564 follow-up MDI treatments were administered, resulting in 1,047 person-hours of therapist time at a labor cost of $14,449. Although the volume of MDIs is only about one fourth that of SVN, the proportionate costs of MDI use are substantially smaller, owing to the 2.6-fold decrease in therapist time required to administer follow-up MDI treatment. For the 12,564 follow-up MDI treatments administered in 1989, the 8-minute/follow-up-treatment savings (compared with labor costs for SVN use) translates to 1,675 person-hours, or a labor cost savings of $23,031 for 1989. Averaged over the year, this time savings translates to 4.5 hours of therapist time/day.

Using the Clinic's direct costs for MDI vs SVN treatments, Table 3 presents the cost impact of MDI use under varying conditions, meant to apply to other institutional settings. For purposes of this analysis, both the total volume of inhaled therapies administered and the proportion of therapies administered by MDI vs SVN are varied, with a net savings associated with MDI use represented (ie, expected costs with all therapies administered by SVN minus actual costs with the proportion of MDI used as indicated in the right-hand columns). For example, in a hospital that provides 150,000 bronchodilator treatments annually, net annual savings associated with administration of all treatments by MDI would be $316,500. If only 20% of bronchodilator treatments were administered by MDI, the annual cost savings would decline to $63,300.

Discussion

Our report demonstrates that on a per-use basis MDI treatment with a spacer device is less expensive than SVN treatments. The principal sources of savings associated with MDI use are the decreased labor costs in follow-up for patients using MDIs and the decreased cost of medications using MDIs with the delivery policy described (ie, single-canister use for multiple patients, each of whom has a spacer device). These two cost savings with MDIs offset the higher equipment cost of MDI use, which is principally associated with the spacer device. Overall, on a per-patient basis (assuming an equivalent duration of therapy with both MDI and SVN), the cost of MDI use is only 44% of the cost of SVN use in our institution. An obvious corollary is that with increasing proportions of MDI use, the net reduction in institutional costs increases. To generalize this analysis to hospitals in which the volume of bronchodilator treatments either falls below or above the 70,000 yearly treatments administered at our hospital, Table 3 demonstrates that the net reduction in costs may be high in hospitals where even moderate numbers of bronchodilator treatments are provided.

Another consequence of increasing MDI use is the savings in therapist time, largely resulting from the shorter time required to supervise MDI use than to administer SVN therapy. In the current analysis, in which MDI use accounted for 18% of all bronchodilator treatments, the associated time savings translates to 4.5 hours of total therapist time/day.

An obvious caution in interpreting a cost analysis from a single institution is that the results may not be generalizable. Three other studies have examined the cost impact of substituting MDIs for SVNs and have also emphasized the cost savings associated with MDI use.[1-3] However, comparison of the analyses is difficult. Jasper et al[4] estimated that substitution of MDIs for SVN treatments in all non-ICU adult patients at Cedars-Sinai Hospital caused a direct cost savings of $253,487/year, based on 47,038 bronchodilator treatments administered. Summer et al[2] reported a threefold reduction in costs associated with using MDI instead of updraft nebulizers,[4] and Berenburg et al[8] reported a minimum of $42,000 annual savings associated with switching to MDIs in a 462-bed hospital. The estimated savings in our institution falls between those previously reported,[4,8] due to the larger size of our institution (approximately 1,000 beds) and the fact that ordering practices in our institution have not yet completely substituted MDI use for SVNs. Specifically, in the second year of the current analysis, MDIs accounted for only 18% of total bronchodilator treatments administered. We believe this ordering practice reflects a persisting (and widely held) misimpression that bronchodilators are more effective when administered by SVN than by MDI.[2] Implementation of our MDI policy (which encourages MDI use when patients

Table 3. Cost Analysis of Patterns of Bronchodilator Therapy: Metered Dose Inhaler (MDI) vs Small Volume Nebulizer (SVN)

Total Therapies Annually	Total Cost of All Therapies by SVN*	Total Cost of All Therapies by MDI	Net Annual Savings When Specific Percentage of Therapies Administered by MDI†				
			100%	80%	60%	40%	20%
20,000	$ 71,400	$ 29,200	$ 42,200	$ 33,760	$ 25,320	$ 16,880	$ 8,440
35,000	$ 124,950	$ 51,100	$ 73,850	$ 59,080	$ 44,310	$ 29,540	$ 14,770
50,000	$ 178,500	$ 73,000	$ 105,500	$ 84,400	$ 63,300	$ 42,200	$ 21,100
75,000	$ 267,750	$ 109,500	$ 158,250	$ 126,600	$ 94,950	$ 63,300	$ 31,650
100,000	$ 357,000	$ 146,000	$ 211,000	$ 168,800	$ 126,600	$ 84,400	$ 42,200
125,000	$ 446,250	$ 182,500	$ 263,750	$ 211,000	$ 158,250	$ 105,500	$ 52,750
150,000	$ 535,500	$ 219,000	$ 316,500	$ 253,200	$ 189,900	$ 126,600	$ 63,300
175,000	$ 624,750	$ 255,500	$ 369,250	$ 295,400	$ 221,550	$ 147,700	$ 73,850
200,000	$ 714,000	$ 292,000	$ 422,000	$ 337,600	$ 253,200	$ 168,800	$ 84,400

* Total cost for SVN and MDI does not include labor cost for initial patient instruction. SVN cost/treatment = $3.57; MDI cost/treatment = $1.46.

† Savings/MDI treatment over SVN treatment = $2.11.

are appropriate candidates and respiratory therapy input is solicited) has been associated with a threefold rise in the proportion of bronchodilators delivered by MDI at our institution, but we aspire to even higher utilization.

In addition to extending previous analyses by examining 2-year trends, the current analysis is based on real-time cost measurements of time expended for respiratory therapy services. Though estimates of time expended can be attained by timing sampled therapies, we believe that using real-time measurement enhances the accuracy of the current analysis.

The differences in the costs of therapy in each analysis are another source of variation in the available savings estimates. Despite institutional variations in the magnitude of savings, there is consensus among available studies that substituting MDIs for SVNs in managing non-ICU adult patients reduces direct costs; and to the extent that MDI therapy is equally effective to SVN therapy, MDI use is preferred. Associated benefits are the savings in therapist time, which can allow more appropriate allocation of respiratory therapy services.[20]

Although our analysis does examine the cost reductions with MDI use when both the volume of total bronchodilator treatments and the proportion of MDI treatments varies (Table 3), obvious impediments exist to direct application of this analysis to other institutional settings. Specifically, estimates of salary costs and medication costs reflect local influences. Also, our analysis averages the costs of the most commonly used MDIs in our institution (salbutamol and ipratropium bromide), which may also differ from other settings. Although it is also true that institutional size, number of therapists available, and the demand for respiratory care services can affect the time estimates for MDI or SVN administration, a strength of this analysis is the reliability of the real-time estimates.

Another impediment to generalization of these results is that the costs associated with labor and medications may change with time. However, to the extent that costs associated with MDI vs SVN change proportionately, the savings associated with MDI use will not change.

In Conclusion

Our analysis demonstrates that delivering bronchodilators by metered dose inhalers with spacers instead of small volume nebulizers in non-ICU adult in-patients is associated with substantial reduction in institutional cost. An impediment to fully realizing these savings is continued dependence on small volume nebulizer treatment for patients who can be equivalently managed with metered dose inhalers with spacer devices. Strategies to enhance metered dose inhaler use may include specific respiratory therapy bronchodilator protocols call-

ing preferentially for metered dose inhalers, and the implementation of a respiratory therapy consult service that allocates respiratory therapy services according to established guidelines.

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Comparison of Tidal Volumes, Minute Ventilation, and Respiratory Frequencies Delivered by Paramedic and Respiratory Care Students with Pocket Mask versus Demand Valve

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BACKGROUND: The importance of establishing the airway and providing ventilation during resuscitation is well recognized; however, the effectiveness of demand-valve ventilation has not been fully addressed. METHODS & MATERIALS: We examined the differences in respiratory frequency (f), tidal volume (V_T), and minute ventilation ($\dot{V}_E$) achieved by 15 emergency medical technicians (EMT) in a paramedic training course (EMT-4) and 16 second-year respiratory care students (RC) when ventilating a mannequin by three different methods: Laerdal pocket mask, a mask designed for mouth-to-mask ventilation (Method I), demand valve with one hand (Method II), and demand valve with two hands (Method III). RESULTS: The RC f was much higher for all three methods compared to the EMT-4. The EMT-4 demonstrated larger V_T for each method compared to the RC. The RC only achieved acceptable V_T with Method III. The EMT-4 and RC demonstrated statistically significant differences in $\dot{V}_E$ between each method. However, the clinical importance of these differences for EMT-4 is negligible. Although the mean values indicated achievement of minimum V_Ts by the EMT-4 with the pocket mask, 40% were unable to do so at 1 minute and 60% at 5 minutes. A great difference was seen in $\dot{V}_E$ produced by RC with Method I at both 1 and 5 minutes vs Methods II and III. CONCLUSIONS: EMT-4 appear to have superior ventilation skills compared to RC. Superior performance skills may be related to (1) different instructional methodologies, (2) effects of various amounts of experience in ventilating actual victims, and (3) greater familiarity of EMTs with the demand valve. As a consequence of this study, we are re-evaluating the instructional methodology used in our programs; we encourage other programs to do the same. We recommend that EMS providers and hospitals consider changing to an acceptable alternate mask in light of these and previous data. (Respir Care 1991;36:1105-1112.)

Background

From the the first publication of *Standards and Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiac Care*,[1] the preeminent place of establishing the airway and breathing (ventilation) in resuscitation has been recognized. In fact, Grauer and Kravitz[2] emphasized that ventilation is second in importance only to defibrillation during resuscitation. In most prehospital care, the patient does not initially have an endotracheal or tracheostomy tube in place, and effective positive-pressure ventilation requires maintaining a seal between the ventilation device and the airway of the patient. In the past, this ventilation has been mouth-to-mouth; however, with the spread of the human immunovirus, or HIV (despite the low likelihood of its transmission during mouth-to-mouth contact),[3] people have begun to realize that a number of infectious diseases can be transmitted via mouth-to-mouth ventilation. Therefore, the American Heart

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Association (AHA) Emergency Cardiac Care Committee (ECC) recommends that a face mask with one-way valve be used by those asked to provide CPR in the course of their employment (eg, emergency medical services [EMS] personnel, police, firefighters, lifeguards, and hospital workers).[3] As an alternative to exhaled-air ventilation, some EMS prehospital-care providers use an operator-triggered, pressure-limited, gas-powered resuscitation device (usually called a demand valve) applied via a mask or an esophageal obturator airway (EOA). When powered by compressed oxygen, these devices have the advantage of delivering 100% oxygen as contrasted with the 16-18% provided by exhaled-air ventilation. However, when the Emergency Care Research Institute (ECRI) tested these devices, it found that each failed to meet at least one widely accepted criterion for performance or patient safety.[4]

Studies and Findings

Many studies have evaluated the ability of various personnel to ventilate patients[5-7] and mannequins[8-15] with manual resuscitators and masks (also called bag-valve-mask) or with mouth-to-mask pocket masks.[5-15] A 1991 paper,[16] published in this journal, demonstrated the effect of oxygen enrichment on oxygen concentration delivered (F_{DO_2}) by mask-to-mouth ventilation. In addition, evaluations of equipment and operator characteristics (such as use of one hand versus two hands to compress the resuscitator bag) have been published.[17-19] However, to our knowledge, no clinical study has addressed the efficacy of ventilation provided by gas-powered resuscitators.

Effectiveness of Mask Ventilation

Elam and colleagues[5] first evaluated the efficacy of mouth-to-mask ventilation in 1954 by having subjects ventilate patients by breathing into the opening of an anesthesia mask. They found that subjects were able to maintain the patient's P_{aCO_2} within normal (and in one subject below normal) limits. In a mask design similar to that in use today, Tomashefski and Oliver[6] added two one-way valves so that the rescuer did not have to remove his mouth from the mouthpiece to inhale. They found this provided safe, effective ventilation of apneic patients. Harrison et al[7] compared ventilation by means of a manual resuscitator attached to an endotracheal tube (as the standard) to that delivered by mouth-to-EOA, manual resuscitator to EOA, manual resuscitator with mask, and mouth-to-pocket mask. They found that tidal volumes (V_{TS}) using the mouth-to-mask method were comparable to those using manual resuscitator-to-endotracheal tube, whereas those achieved using manual resuscitator with mask were less than half those achieved with the manual resuscitator and endotracheal tube. They concluded that mouth-to-pocket mask was the preferred means of initial ventilation. Subsequently, Elling and Politis[8] evaluated the ability of EMTs to ventilate a mannequin with five different manual resuscitators (all using the same mask) and mouth-to-mask technique. Average V_T with mouth-to-pocket mask was 999 mL; with manual resuscitators as a group, it was 641 mL. Reasons cited for the difference were difficulty in maintaining mask seal with one hand and delivery of smaller V_T from the manual resuscitator when only one hand was used. They recommended using either a mouth-to-pocket-mask or a manual resuscitator with one person holding the mask and a second person compressing the bag. Lawrence and Sivaneswaran[11] found V_{TS} delivered mouth-to-mouth were acceptable even before instruction in proper technique and those delivered by mouth-to-mask were acceptable after instruction. Only 2 out of their group of 70 were able to achieve adequate (> 800 mL) V_{TS} using bag-valve-mask technique. After instruction, V_{TS} delivered with a Robertshaw gas-powered resuscitator were more than adequate (an average of 1,370 mL).

The first pocket mask on the market was one manufactured by Laerdȧl that did not allow oxygen enrichment. Safar modified the original mask by adding an oxygen inlet nipple with a one-way valve.[12] This allowed exhaled-air ventilation with an increased F_{IO_2} or positive-pressure ventilation by intermittent occlusion of the mask port. Sivaneswaran and colleagues[13] designed their own mask for mouth-to-mask ventilation, adding an oxygen inlet valve but also placing a mouthpiece on the mask to facilitate sealing the rescuer's lips. Masks

of many different designs have appeared on the market in recent years. Hess and colleagues evaluated 8 different masks and found that subjects were able to average V_Ts less than 0.70 L with 3 of them; V_Ts were greater than 0.75 L with the other 5.[14] The differences in the volumes were related to how readily the rescuer could seal the mask to the victim's face. Stewart et al[15] had similarly evaluated mask design as a factor in ventilation but with a manual resuscitator as the gas source. They found that the SealEasy mask afforded an easier seal than did an old-design Laerdal mask or a Robertshaw anesthesia mask.

Sainsbury's group[20] tested the ability of 100 nurses to ventilate a mannequin by 6 methods: mouth-to-mouth, mouth-to-face mask, RM-1 and Mistviva (two different demand valves), Airviva, and Mapleson bag at 14 L/min oxygen flow. They defined 'passing' as delivering at least a 6-L minute volume ($\dot{V}_E$) and found pass rates of 88, 66, 55, 52, 25, and 15%, respectively.

Although operator-triggered, gas-powered resuscitators are in fairly common use, evaluations in the literature are infrequent, at best. Harries[21] simply described those units that were available and stated the characteristics he considered to be ideal in a ventilation device. Osborn and colleagues[22] detailed three case reports of barotrauma inflicted by demand valves that, on testing, turned out to deliver either excess pressure, excess flow, or both. Phillips and Skowronski[23] evaluated a number of different types of ventilation devices but only one demand valve (Robertshaw); their conclusions were essentially the same as those of Osborn et al.[22]

The dearth of literature on the clinical performance of demand valves (ie, performance under conditions simulating real life) encouraged us to determine whether there is a difference over time among the V_T, respiratory frequency (f), and $\dot{V}_E$ delivered by two different groups of rescuers to a mannequin with a Laerdal pocket mask (ie, mask designed for mouth-to-mask ventilation), demand valve using one hand to seal the mask, and demand valve using two hands to seal the mask. The null hypothesis was that no statistically significant difference exists in tidal volume, respiratory frequency, and minute ventilation provided by the three different methods or between the two groups.

Methods and Materials

Study Subjects

Fifteen students (11 men, 4 women) all of whom were certified EMTs with various amounts of field EMS-experience and who were enrolled in a paramedic course at our institution (EMT-4) and 16 second-year respiratory care students (7 men, 9 women) from our associate degree and baccalaureate programs in respiratory care (RC) comprised the subjects for this study. All subjects had current completion cards for the American Heart Association's Provider Course C (Professional Rescuer) in Basic Cardiac Life Support. None of the subjects had taken the American Heart Association's Advanced Cardiac Life Support Course. All of the EMT-4 subjects were members of volunteer ambulance or rescue squads and had been EMTs for varying periods of time. The RC students had 18 months of clinical experience in our program and, at most, 6 months part-time experience working as staff therapist or technician. Before participating in the study, the subjects read and signed a consent form, as required by our Institutional Review Board for the Protection of Human Subjects.

Study Protocol

All subjects were instructed to ventilate a Laerdal Resusci-Anne Recording mannequin* for 5 minutes at the appropriate f and V_T. The mannequin had been placed on the floor to simulate an actual rescue situation that both pre- and in-hospital care providers were likely to encounter. The subjects ventilated the mannequin each of three ways with the sequence chosen randomly by having the subject select 1 of 6 cards on which the 3 numbers corresponding to the 3 methods were listed. The methods were Method I—pocket mask, Method II—demand valve holding the mask with one hand, and Method III—demand valve holding the mask with two hands (using the trigger lever). The sub-

Suppliers are identified in the Product Sources section at the end of the text.

jects ventilated the mannequin for 5 minutes by each method, with a 10-minute rest between methods. The 5-minute time period was chosen as a convenient period for measurement that would also induce some fatigue. All subjects were observed by one of the two authors. The same mannequin was used for all trials. The same demand valve, mask, and pocket mask were used throughout the study. However, each participant received his own one-way valve for pocket-mask trials. Ventilation was measured during the first and last minute of each trial with a Bourns VM-90 spirometer attached to the exhalation port on the mannequin; $\dot{V}_E$ and f were recorded. The subject was unable to see either the volume-indicating device on the mannequin or the display of the VM-90.

Equipment Preparation and Calibration

Prior to each experimental session, the line pressure of the piped oxygen supply used to power the demand valve was checked to ensure that it met the specifications of the demand-valve manufacturer. The VM-90 was checked for calibration with a 1-L calibration syringe to ensure that it met the stated accuracy of ± 5%. To confirm that there were no leaks in the mannequin system, the recorder was turned on, the outlet port was occluded, and a breath was delivered. The recorder was allowed to run for a few seconds, and the recording was then checked for the presence of a plateau, indicating no volume loss.

Mean and standard deviation for V_T, f, and $\dot{V}_E$ were calculated for each subject for each technique. Data were to be analyzed by a commercially available statistical package using two-factor repeated measures of analysis of variance (ANOVA). We considered that statistical significance would be achieved at $p \leq$ to 0.05. The two factors (independent variables) were method and group. The repeated measure was time; measurements were taken at the end of 1 and 5 minutes. In addition, Tukey post-hoc comparisons was performed across time by method and group.

Results

Table 1 provides the range, mean, and standard deviation values for V_T, f, and $\dot{V}_E$ by method and time for the two groups of students. Table 2 pro-

Table 1. Comparison of AHA-Recommended Tidal Volumes, Minute Ventilation, and Frequencies to Those Obtained by Emergency Medical Technician Students (EMT-4) and Respiratory Care Students (RC) by Three Methods

Variables	AHA Values	Method I		Method II		Method III	
		EMT-4	RC	EMT-4	RC	EMT-4	RC
Tidal Volume (L)							
Range at 1 min	_	0.44-1.81	0.24-0.74	0.64-1.32	0.33-1.41	0.61-1.46	0.38-1.47
Mean (SD) at 1 min	0.8 (0)	0.90 (0.35)	0.39 (0.14)	0.98 (0.25)	0.77 (0.33)	1.03 (0.27)	0.90 (0.34)
Range at 5 min	_	0.34-1.65	0.11-0.91	0.39-1.28	0.27-1.30	0.69-1.31	0.34-1.43
Mean (SD) at 5 min	0.8 (0)	0.79 (0.37)	0.36 (0.24)	0.96 (0.30)	0.75 (0.34)	0.96 (0.17)	0.85 (0.30)
Frequency (breaths/min)							
Range at 1 min	_	6-20	10-37	9-20	14-34	8-18	16-33
Mean (SD) at 1 min	12 (0)	12.0 (3.53)	19.8 (7.12)	13.5 (3.10)	23.1 (6.41)	13.70 (2.70)	23.3 (5.08)
Range at 5 min	_	6-35	11-32	7-19	10-34	7-24	14-37
Mean (SD) at 5 min	12 (0)	12.5 (6.83)	19.2 (5.71)	12.3 (3.77)	20.6 (6.70)	12.7 (4.15)	22.6 (6.81)
Minute Ventilation (L)							
Range at 1 min	_	4.9-19.9	3.60-14.7	6.4-23.8	5.58-31.0	9.6-19.0	7.61-32.3
Mean (SD) at 1 min	9.6 (0)	10.52 (4.44)	7.46 (2.97)	13.22 (4.55)	17.71 (8.42)	13.7 (3.01)	20.39 (7.09)
Range at 5 min	_	2.9-18.7	1.36-17.3	4.7-24.3	5.92-28.7	7.6-24.6	5.12-31.30
Mean (SD) at 5 min	9.6 (0)	9.18 (4.59)	6.83 (4.57)	11.66 (5.08)	14.86 (7.56)	12.19 (4.47)	18.75 (6.99)

Table 2. Tukey Post-Hoc Comparisons of Means across Time by Method and Group for Pocket Mask (Method I) Demand Valve with One Hand (Method II), and Demand Valve with Two Hands (Method III) for EMT-4 Students and Respiratory Care Students (RC)

Method and Group	Mean Difference
Minute Ventilation ($p \leq 0.05$ for mean differences ≥ 6.130)	
Method III/RC vs Method III/EMT-4	6.608
Method III/RC vs Method I/RC	12.426
Method II/RC vs Method I/RC	9.142
Tidal Volume ($p \leq 0.05$ for mean differences ≥ 0.344)	
Method III/RC vs Method I/RC	0.499
Method II/RC vs Method I/RC	0.385
Respiratory Frequency ($p \leq 0.05$ for mean differences ≥ 1.509)	
EMT-4 students vs RC students	8.639
Method II vs Method I	1.533

vides the results of the Tukey post-hoc comparisons for V_T, f, and $\dot{V}_E$.

Respiratory Frequency

The ventilation rates produced by the RC students were higher by all three methods than those produced by the EMT-4 students (for means $p < 0.001$, for method $p = 0.003$), and the post-hoc comparisons (Table 2) show that differences between the two groups are statistically significant for all three methods.

Tidal Volume

For tidal volumes, differences in means for group ($p = 0.001$), method ($p < 0.001$), group by method ($p = 0.011$), and time ($p = 0.022$) were significant. The EMT-4 students delivered more consistent and larger V_Ts for each method as a group when compared to the RC students; they were able to achieve adequate mean V_Ts (≥ 800 mL) with the pocket mask at 1 minute and slightly less than (by 0.01 L) at 5 minutes. What these means obscure, however, is the fact that 9 of 15 EMT-4 students were unable to achieve adequate V_T at 5 minutes and 6 were unable at 1 minute also (Figs. 1 & 2). As a group, the RC students demonstrated marked

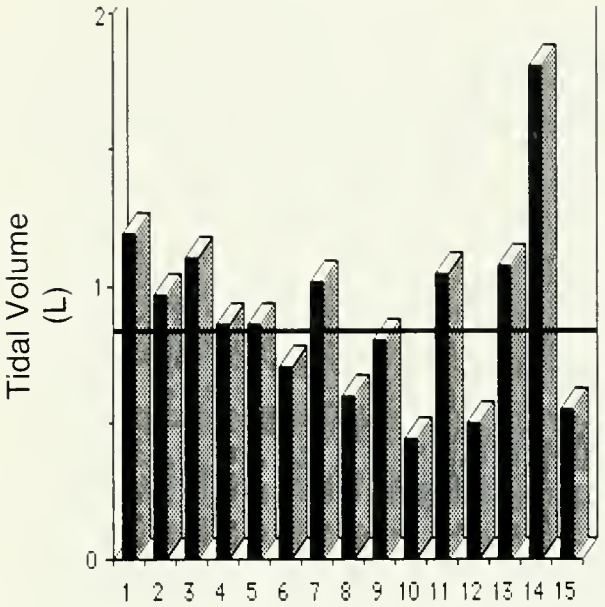

Fig. 1. Tidal volume produced by 15 emergency medical technicians in a paramedic course at 1 minute with pocket mask.

differences in V_T among the methods, particularly Method I and II and III. The post-hoc comparison confirms a statistically significant difference between the V_T delivered by Method I and both Method II and III. In addition, the RC students as a group did not achieve adequate V_T with either Method I or Method II. In comparing the two groups, it is interesting to note that the RC students achieved V_Ts comparable to the EMT-4 only when

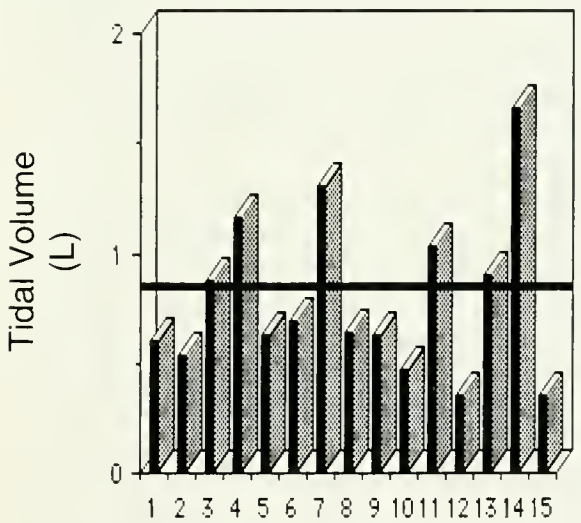

Fig. 2. Tidal volume produced by 15 emergency medical technicians in a paramedic course at 5 minutes with pocket mask.

achieved V_{TS} comparable to the EMT-4 only when using Method III.

Minute Ventilation

There was a statistically significant difference in the means for method ($p < 0.001$), group by method ($p < 0.001$), and time ($p < 0.001$) between the EMT-4 students and RC students. There were no statistically significant differences seen when comparisons (using ANOVA) were made of group by method, method by time, and group by method by time. However, post-hoc comparison revealed a statistically significant difference between EMT-4 and RC for Method III. There also was a statistically significant difference for RC comparing Method I with Method II and Method I with Method III. There appears to be a great difference in the $\dot{V}_E$ produced by the EMT-4 students versus the RC students, but the standard deviations are so large that statistical significance is not achieved. Both the EMT-4 students and RC students demonstrated statistically significant differences in $\dot{V}_E$ among the methods, although the clinical importance of these differences for the EMT-4 students would seem to be negligible. A great difference was seen for the RC students in $\dot{V}_E$ produced by Method I at both 1 and 5 minutes versus Methods II and III.

Discussion

Although Hess and colleagues[14] found that rescuers produced V_{TS} less than 800 mL using the Laerdal pocket mask, this device is commonly used both in prehospital EMS and also in hospitals in our area.

The EMT-4 students were clearly superior to the RC students in terms of producing acceptable f, $\dot{V}_E$, and V_T for most methods at both 1 and 5 minute measurements. If we assume a $\dot{V}_E$ of 9.6 L to be minimally acceptable by AHA criteria (V_T 800 mL, f 12/min),[24] then the EMT-4 students were able to achieve minimum $\dot{V}_E$ by every method and at both times except by Method I at 5 minutes (and then only missing the minimum level by 0.42 L). The RC students were unable to achieve minimum $\dot{V}_E$ by Method I; they did achieve acceptable ventilation by both Methods II and III only by virtue of their very high ventilation rate. There was a clock with a sweep second hand at a 90° angle to the subjects' line of sight. This may have aided the EMT-4 students in delivering the appropriate respiratory rate, although it appears to have been ignored by the RC students. It may also have allowed each group to deliver consistent rates.

There was a downward trend with time seen in all trials for all three methods for V_T and $\dot{V}_E$. The cause for this drop may have been fatigue. This trend was not unexpected, but we question the actual clinical importance of the trend during the measurement period, except in those instances where the rescuer was not initially achieving even a minimally acceptable V_T and/or $\dot{V}_E$. This occurred for EMT-4 students at 5 minutes for Method I with respect to V_T and $\dot{V}_E$ and the RC students at 1 and 5 minutes with respect to V_T for Methods I and II, and at 1 and 5 minutes for Method I with respect to $\dot{V}_E$. An additional factor to be considered is practice or learning during the time period, which would tend to improve performance.

The major problem detected here is that the further fall in V_T, even in the face of an apparently acceptable $\dot{V}_E$, would result in an increase in the dead-space-to-tidal-volume ratio (V_D/V_T) and thus an actual fall in minute alveolar ventilation. Also, were the downward trend to continue beyond the 5-minute measurement period, the result, at some point, would be the delivery of unacceptably low V_{TS} and unacceptably low alveolar ventilation.

The EMT-4 students appear to have superior psychomotor skills in these basic methods of ventilation. When one looks at individual numbers, one sees that nearly every EMT-4 student was able to achieve adequate and similar tidal and minute volume with the demand valve with both one hand and two hands. A possible explanation for this is the fact that they are all New York State Certified Basic Emergency Medical Technicians with various amounts of field experience. Therefore, there are three possible factors to explain the differences seen in this study: (1) different methods of teaching and practice of these skills, (2) greater familiarity of EMTs with the demand valve, and (3) EMTs' various amounts of experience in venti-lating actual victims.

We believe that the results of this study have important implications for our program and possibly

teaching and practice of these methods of artificial ventilation. We seem to have discovered a defect in a basic but very important part of our curriculum. We will be re-evaluating the methods we use to teach these skills as well the amount of time allotted to practice and remediate these skills; we encourage other programs to do the same. (It is interesting to note that the demand valve appeared in the latest RRT exam matrix, especially in view of its apparently limited use in hospitals.)

In Conclusion

Our study confirms the results of Hess et al[14] regarding the inability of rescuers to deliver adequate V_Ts with the Laerdal pocket mask, in spite of the misleadingly high mean values seen. We recommend that prehospital EMS providers and hospitals change to one of the masks identified by Hess as allowing ready delivery of adequate V_Ts. In addition to the findings of this study, we have identified four topics for further research: (1) effect(s) of fatigue on psychomotor performance over longer periods of time, (2) difference(s) in the teaching and practice between the EMT-4 courses and the Respiratory Care Program curriculum, (3) effect(s) of field experience on psychomotor performance, and (4) change in performance due to learning during the time of performance of the skill.

PRODUCT SOURCES

Demand Valve:
Model 4201, Robertshaw Controls, Anaheim CA

Pocket Mask:
Catalog number 83 00 03, Laerdal Medical Corp, Armonk NY

Resuscitation Mannequin:
Recording Resusci-Anne, catalog number 20 00 00, Laerdal Medical Corp, Armonk NY

Ventilation Monitor:
VM-90, Bear Medical Systems, Riverside CA

1-Liter Calibration Syringe:
Warren E Collins Inc, Braintree MA

Stop Watch:
Heuer-Leonides SA, Switzerland

Statistical Software:
SYSTAT $_{TM}$, Evanston IL

ACKNOWLEDGMENTS

The authors thank Douglas U Smith PhD for his assistance with the statistical analysis and Richard Cherry for his assistance in recruiting paramedic students for the study.

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Compression Volume in Adult Ventilator Circuits: A Comparison of Five Disposable Circuits and a Nondisposable Circuit

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Although the concept of ventilator circuit compression is well known, it is not fully appreciated clinically. We compared the compression volume of five adult disposable ventilator circuits and a nondisposable circuit. METHODS: Five brands of disposable circuits (Inspiron, Intertech, Marquest, Seamless, and U-Mid) and one nondisposable brand (Bennett) were used. The circuits were attached to the outlet of a Bennett MA-1 ventilator in the standard manner, and the filter and humidifier were bypassed to eliminate their contribution to compression volume. The ventilator delivered 1 L of gas to a Michigan Instruments Test Training Lung at a flow of 60 L/min and a rate of 12/min. A valve system was placed between the Y-connector and the TTL to partition circuit compression volume from the volume delivered to the test lung. The compression factor was calculated by dividing the compression volume by the ventilating pressure. Five compliance settings were used on the TTL (0.1, 0.05, 0.03, 0.02, and 0.015 L/cm) to simulate different ventilating pressures. Pressure at the Y-connector, compressible volume, and volume delivered to the TTL were measured using a calibrated Timeter RT200 volume-pressure analyzer. Five measurements were made with each brand. A new circuit was used for each run with a disposable circuit, and a different circuit from the respiratory care department stock was used for each run with a nondisposable circuit. RESULTS: There were significant differences between the compression factors at different TTL compliance settings ($p < 0.001$), and brands of circuits evaluated ($p < 0.001$). There was a significant interaction effect for compression factors between TTL compliance setting and circuit brands ($p < 0.001$). The compression factors for all disposable circuits were greater than the compression factors for the nondisposable circuits ($p < 0.05$). CONCLUSIONS: There were differences in circuit compression factors as a function of the ventilating pressure and the brand of circuit. Although these differences are too small to be clinically important in many cases, they may be important during ventilation with low tidal volumes and high pressures. (Respir Care 1991;36:1113-1118.)

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A version of this paper was presented by Mr Simmons at the RESPIRATORY CARE OPEN FORUM during the 1990 AARC Annual Meeting in New Orleans, Louisiana.

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Introduction

The concept of compression volume in ventilator systems is well known. However, it is often not fully appreciated in the clinical setting, and it has been the subject of very little scientific inquiry.[1-4] Compression volume is the result of the internal volume of the ventilator, the volume of the in-line humidifier, and the characteristics of the circuit tubing. Although the circuit compression volume does not reach the patient, and thus decreases the delivered volume, it is measured with the exhaled gases from the patient. The compression volume of the circuit tubing is a function of the volume of the circuit, the compliance of the tubing

material, and the ventilation pressure. The volume of gas compressed in the ventilator circuit becomes clinically important when high ventilation pressures are required, particularly if the tidal volume is small. It affects calculations of lung mechanics[5] and auto-PEEP.[6] Many hospitals use disposable ventilator circuits, which are available from a number of manufacturers. The purpose of this study was to evaluate the compression volume of five disposable ventilator circuits and a nondisposable circuit.

Methods

Five brands of disposable circuits were evaluated: Inspiron 003291 Universal Volume Ventilator Circuits, Intertech 003280 Universal Volume Ventilator Circuits, Marquest 157514 Universal Volume Ventilator Circuits, Seamless 5844 Universal Ventilator Sets, and Becton Dickinson 5060 U-Mid Moduflex Manifold Sets.* These circuits were chosen because they are commonly used in our area and were readily available from a local respiratory care equipment vendor. Nondisposable Bennett circuits (#4-009864-00) were also evaluated. The disposable circuits were new and were used as supplied from the manufacturer. The nondisposable circuits were chosen at random from the respiratory therapy department stock.

Circuits were attached directly to the outlet of a Bennett MA-1 ventilator. Because we were interested in evaluation of the compression volume of the circuit tubing only, we chose not to include a humidifier or filter in the circuit. Ventilation volumes were delivered to a Michigan Instruments TTL test lung. The ventilator was set to deliver breaths at a rate of 12/min, a flow of 60 L/min, and a volume of 1.0 L; these were measured using a Timeter RT200 volume-pressure analyzer. The RT200 analyzer was calibrated according to manufacturer's specifications, its volume accuracy was confirmed using a 1.0-L calibration syringe, and its pressure accuracy was confirmed using a mercury reservoir manometer. All measurements were made at atmospheric temperature and pressure (ATP) conditions.

*Suppliers are identified in the Product Sources section at the end of the text.

An Instrumentation Industries RI 27-142 valve system was placed between the Y-connector and TTL to partition gas compressed in the circuit from gas delivered into the test lung (Fig. 1). Pressure at the Y-connector, circuit compressed volume, and volume delivered to the TTL were measured using the RT200. The compression factor was calculated by dividing the compression volume by the corresponding ventilation pressure.

Five compliance settings were used on the TTL to produce different ventilation pressures: 0.1, 0.05, 0.03, 0.02, and 0.015 cm H_2O. This resulted in ventilation pressures of 19, 27, 38, 51, and 63 cm H_2O, respectively. Five measurements were made with each circuit brand, using a different circuit for each set of TTL compliance settings. Mean and standard deviation (SD) were calculated for the compression factor of each circuit brand at each TTL compliance setting. Two-way analysis of variance was used to compare compression factors, using TTL compliance setting as a repeated measures (within groups) factor, and circuit brand as a grouping (between groups) factor. A Scheffe post-hoc analysis was performed to identify significant differences between levels within a factor. All statistical analysis was performed using standard methodology[7] and a commercially available computerized software package.

Results

The data are summarized in Table 1 and Figures 2-7. There were significant differences between the compression factors at different TTL compliance settings ($p < 0.001$) and brands of circuits evaluated ($p < 0.001$). The compression factors were greater for all disposable circuits than the compression factors for the Bennett circuits ($p < 0.05$). The compression factors at low compliance settings on the TTL (high ventilating pressures) were greater than the compression factors at high compliance settings (low ventilating pressures) ($p < 0.05$). However, there was a significant interaction effect for compression factors between TTL compliance setting and circuit brands ($p < 0.001$), meaning that the relationship between compression factors and compliance settings was not consistent among the circuits evaluated; this can be seen in Figures 2-7.

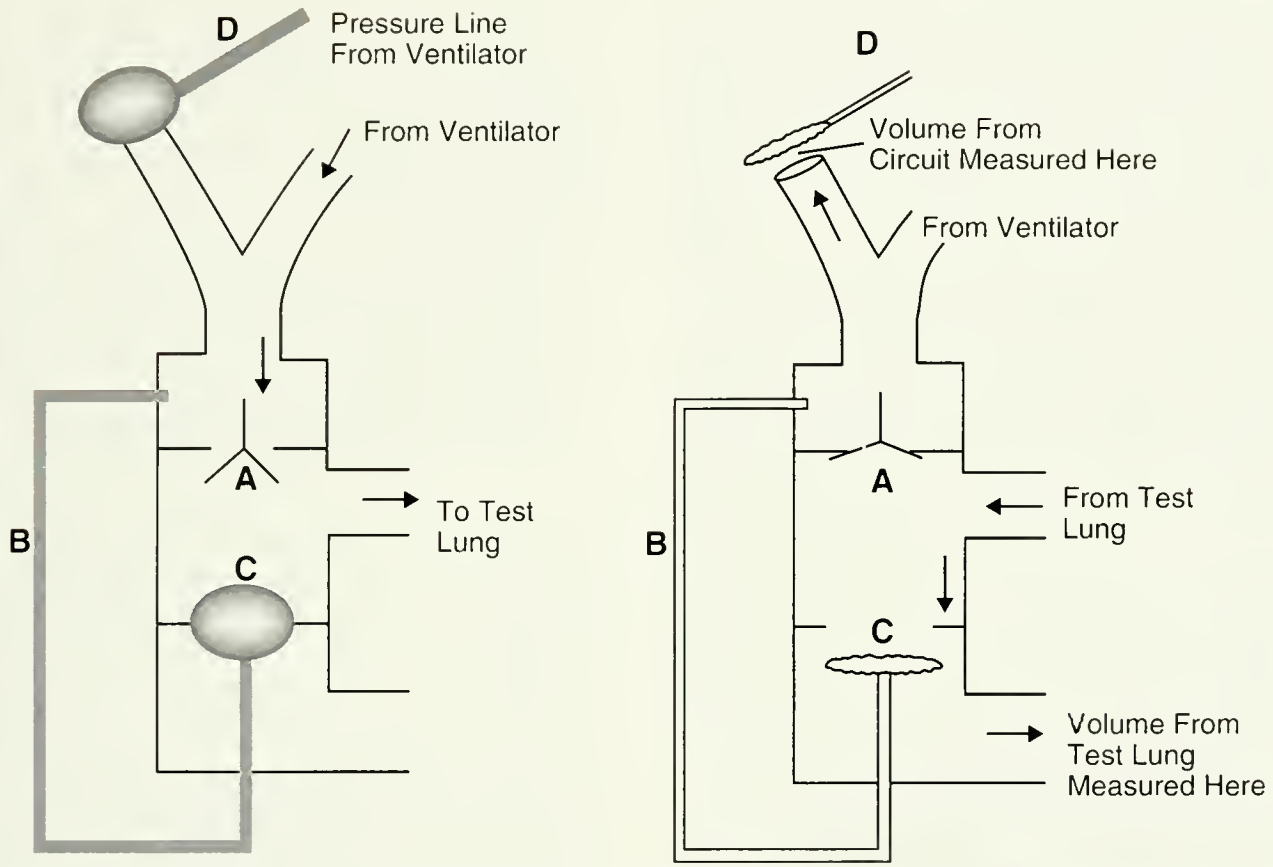

Fig. 1. Schematic illustration of the valve-system used to separate the circuit compression volume from the volume delivered to the test lung. A—one-way valve that opens during inhalation and closes during exhalation; B—hollow tubing to inflate mushroom valve during inhalation; C—mushroom valve that closes during inhalation and opens during exhalation; D—circuit mushroom valve that closes during inhalation. Circuit compression volume is the volume of gas measured from the circuit, and volume delivered to the test lung is the volume of gas measured from the test lung.

Table 1. Compression Factors for the Ventilator Circuits Evaluated, at the TTL Compliance Settings Used

	TTL Compliance Settings (L/cm H_2O)				
Circuit (Part No.)	0.1	0.05	0.03	0.02	0.015
Inspiron (003291)	1.5 (0.03)*	1.6 (0.2)	1.8 (0.1)	2.1 (0.02)	1.9 (0.1)
Intertech (003280)	2.3 (0.3)	2.4 (0.2)	2.5 (0.1)	2.5 (0.1)	2.6 (0.0)
Marquest (157514)	1.4 (0.3)	1.9 (0.1)	2.0 (0.2)	2.1 (0.1)	2.1 (0.0)
Seamless (5844)	1.7 (0.2)	2.1 (0.2)	2.0 (0.1)	2.2 (0.0)	2.0 (0.1)
U-Mid (5060)	1.8 (0.3)	2.0 (0.2)	2.1 (0.1)	2.2 (0.01)	2.2 (0.1)
Bennett (4-009864-00)	1.6 (0.1)	1.5 (0.1)	1.4 (0.1)	1.4 (0.2)	1.4 (0.1)

*Factors are mean (SD) in mL/cm H_2O.

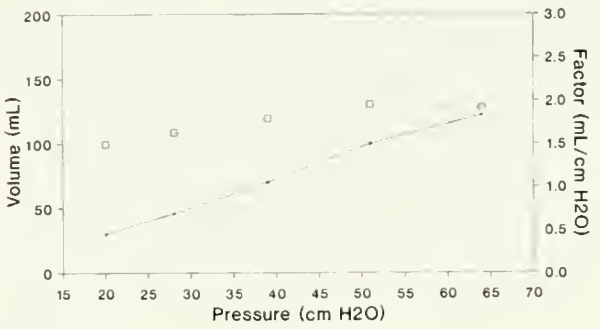

Fig. 2. Compression volumes (—■—) and compression factors (-- □ --) for the Inspiron circuit (#003291).

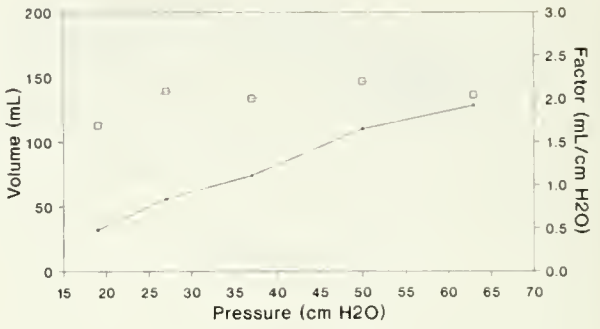

Fig. 3. Compression volumes (—■—) and compression factors (-- □ --) for the Seamless circuit (#5844).

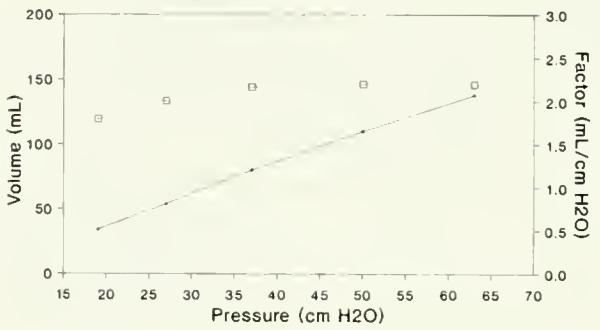

Fig. 4. Compression volumes (—■—) and compression factors (-- □ --) for the U-Mid Moduflex circuit (#5060).

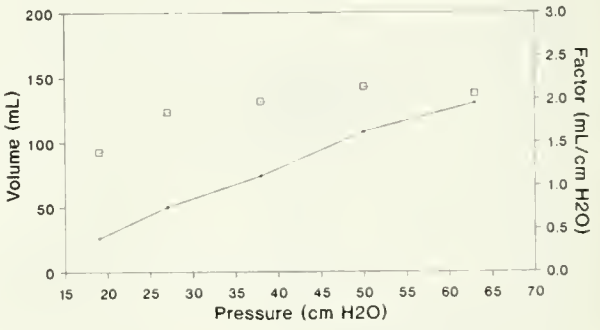

Fig. 5. Compression volumes (—■—) and compression factors (-- □ --) for the Marquest circuit (#157514).

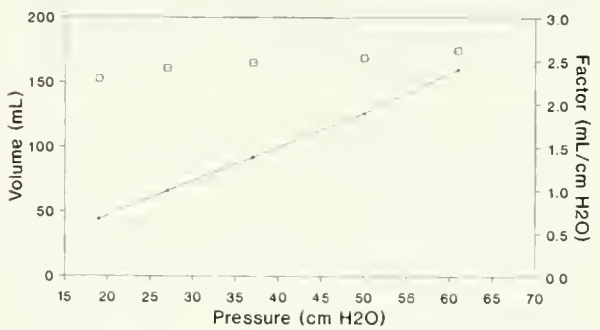

Fig. 6. Compression volumes (—■—) and compression factors (-- □ --) for the Intertech circuit (#003280).

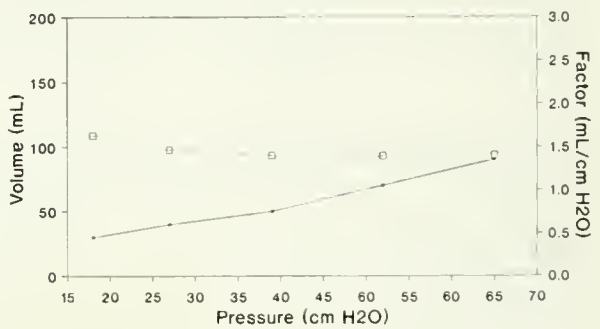

Fig. 7. Compression volumes (—■—) and compression factors (-- □ --) for the Bennett circuit (#4-009864-00).

Discussion

These results show that there are measurable differences in compression factors among commercially available ventilator circuits. Generally, the compression factors of the disposable circuits were greater than those of the nondisposable circuits. This became most pronounced at the lowest TTL compliance setting (highest ventilation pressure); under these conditions, the mean difference between the nondisposable circuits and the disposable circuits was as great as 1.2 mL/cm H_2O (Bennett and Intertech at lowest TTL compliance setting)—a volume of 76 mL. This may be clin-

ically important during ventilation at low tidal volumes and high ventilation pressures.[8]

An unexpected finding was the curvilinear relationship between the compression factor and ventilation pressures (TTL compliance settings) with the disposable circuits. The compression factors tended to be smaller at lower ventilation pressures. We believe that this is because the compression factor at lower pressures is the result of stretch of the circuit material, as well as compression of gas within the circuit. At higher pressures, the limits of distention of the circuit are approached, so that the measured compression factor is primarily due to compression of gases within the circuit.

The differences between compression factors when comparing the highest and lowest TTL compliance settings were as great as 0.7 mL/cm H_2O (Marquest), which would result in an overestimation of compression volume by 13 mL at the higher TTL compliance setting, or an underestimation of compression volume by 44 mL at the lower TTL compliance setting if only one or the other compression factor were used. It is of interest to note that the compression factors for the nondisposable circuit (Bennett) were virtually constant over the range of pressures used in this study; the reasons for this are unclear, but may relate to the characteristics of the material used to construct this circuit.

We did not attempt to measure the compression factors of all commercially available ventilator circuits. Our intent was to determine whether differences exist among brands of circuits, and whether there are differences between disposable and nondisposable circuits. Further work is needed to determine the compression factors of other circuit brands. We made all our measurements at ATP. Further work is also needed to determine whether compression factors are affected by temperature, as can occur in clinical practice. The compression factors that we determined are only for the circuit tubing; in practice, a humidifier is usually placed inline, which further increases the compression volume. Additional work is needed to evaluate the compression factors of commercially available humidifiers such as those used with mechanical ventilators.

Several other studies have evaluated compression volume. Bartel et al[1] compared the compression factors of four ventilator circuits used on four ventilators, with and without use of an inspiratory pause. They found differences in compression factors among circuits, ventilators, and TTL compliance settings; however, an inspiratory pause did not affect compression volume. Bartel et al found a curvilinear relationship between compression factors and ventilation pressures as we did in this study. Forbat and Her[2] evaluated the compression volume of Bennett nondisposable circuits, using methodology similar to that later used by Bartel et al and by us. Although we did not include a humidifier in the circuits that we evaluated, Bartel et al used an empty Bennett Cascade humidifier, and Forbat and Her used a filled Bennett Cascade humidifier. Compression volume has also been evaluated in anesthesia ventilator circuits.[3,4]

Volume is compressed within the bellows of the Bennett MA-1 ventilator during inspiration. However, a one-way valve between the bellows and the circuit closes at end-inspiration, so that any gas compressed in the bellows during inspiration decompresses when the bellows refills during the expiratory phase. Thus, the compression volumes that we measured were due only to the circuits, and were not affected by the compression of gases within the ventilator.[9]

Consideration of compression volume is important for several reasons. Most importantly, compression volume decreases the delivered tidal volume to the patient. As described by Demers et al,[5] compression volume should be considered when lung compliance is estimated in ventilated patients. Scott et al[6] found that auto-PEEP levels were lower if a circuit with low compression volume was used. Although it has been suggested that compression volume affects measurements of oxygen consumption and carbon dioxide production,[1] this has been questioned by others.[10] Compression volume has also been shown to affect measurements of dead space.[2,11] When compression volume is likely to affect physiologic measurements, it may be desirable to use a valve-system to separate the patient's exhaled gases from the gas compressed in the circuit.[1,2]

Conclusions

Ventilator circuit compression factors differ as functions of the circuit brand and the pressure with-

in the circuit. Although these differences are too small to be clinically important in many cases, they should be considered during ventilation with small tidal volumes and high pressures. When compression volume is likely to affect physiologic measurements, a valve-system should be used to separate the patient's exhaled gases from the gas compressed in the circuit.

ACKNOWLEDGMENTS

We thank Breathing Services, Ephrata, Pennsylvania, for their donation of the disposable circuits. We also thank the Pulmonary Services Department, York Hospital, York, Pennsylvania, for allowing us to use Bennett circuits from their stock.

PRODUCT SOURCES

Ventilator Circuits:
003291 Universal, Inspiron, Div of Intertech Resources, Bannockburn IL
003280 Universal, Inspiron, Div of Intertech Resources, Bannockburn IL
5844 Universal, Seamless, Div of Professional Medical Products, Ocala FL
157514 Universal, Marquest Medical Products, Englewood CO
5060 U-Mid Moduflex, Becton-Dickinson Respiratory Systems, Lincoln Park NJ
4-009864-00, Puritan-Bennett Corp, Overland Park KS

Ventilator:
MA-1, Puritan-Bennett Corp, Overland Park KS

Test Lung:
TTL, Michigan Instruments, Grand Rapids MI

Volume-Pressure Analyzer:
RT200, Timeter, Lancaster PA

Valve:
RI 27-142, Instrumentation Industries, Bethel Park PA

Mercury Manometer:
WA Baum Co, Copiague NY

Calibration Syringe:
Vacumed, Ventura CA

Statistical Software:
SPSS/PC+, SPSS, Chicago IL

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A Comparison of the Puritan-Bennett 7200a Ventilator's Flow-By Mode to the T-Piece Mode prior to Extubation in Postsurgical Patients

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We compared flow-by ventilation (FB) via the Puritan-Bennett 7200a ventilator with T-piece ventilation (TP) during weaning from mechanical ventilation (MV). METHODS: We placed 22 consecutive postsurgical patients being weaned from MV on FB at base flows of 10 L/min and 20 L/min and then on TP. Blood pressure, pulse rate, respiratory rate, blood gases, tidal volume, and peak inspiratory flow were measured after at least 20 min in each mode. Statistical analysis of clinical status used a three-level, one-way analysis of variance with technique as a within-subjects factor. Setup costs of the three ventilatory modes were evaluated using relative value units for labor plus actual costs of added equipment and supplies. RESULTS: Although there was a statistically significant difference in P_{aCO_2} among the ventilatory modes, this was not clinically important. No other differences were found. Each FB mode cost $2.55 to set up, whereas TP cost $11.90. CONCLUSIONS: FB and TP were clinically equivalent. However, the alarm and monitoring capabilities during FB are useful and may be worth the one-time cost ($1,000) of adding the optional flow-by software to the 7200a ventilator. (Respir Care 1991;36:1119-1122.)

Background

The process of removing a patient from mechanical ventilation (weaning) usually proceeds along a defined plan of gradual withdrawal from mechanical support. Prior to extubation, a patient is often given a trial of spontaneous breathing through a valveless T-piece system (TP) that allows continuous gas flow with minimal resistance to breathing. This eliminates the resistive qualities of CPAP systems currently found on ventilators that employ a demand-flow system. These resistive qualities can impose a significant ventilatory work load on patients.[1] This increased work load can result in inaccurate assessment of a patient's ability to ventilate independently, or it can even cause the patient to go into respiratory failure.[2-4]

Besides the usual T-piece system, another method is available that mimics continuous-flow systems; this is the optional flow-by (FB) mode on the Puritan-Bennett 7200a ventilator.

The 7200a Ventilator's Flow-By Mode

Flow-by is a software option for P-B 7200a ventilators that are equipped with an internal

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exhalation valve.[5] FB causes a predetermined flow of gas from a base flow of 5 to 20 L/min to be delivered into the patient circuit prior to inspiration, and it vents excess flow through the exhalation valve. Thus, gas is available to the patient as soon as an inspiratory effort is detected by the flowsensor, which responds to a drop in the base flow. The operator must select the base flow and flow sensitivity to set up FB. As the patient begins to exhale, base flow is reduced to 5 L/min, which decreases resistance to the exhaled flow. On detection of the next inspiratory effort, base flow is increased to the preset level.

We evaluated the efficacy of FB as a substitute for TP prior to extubation in our surgical intensive care unit (SICU).

Patients and Methods

During a 2-month period, we collected data on 22 consecutive patients in our SICU who required mechanical ventilation postoperatively, and we also reviewed their charts. Mechanical ventilation was required for a number of reasons (eg, anesthesia reversal, adult respiratory distress syndrome, ventilatory failure). The patients ranged in age from 28 to 95 years, 19 of them were men, and 3 were women. The operative procedures were neurosurgical, thoracic, abdominal, orthopedic, or combinations thereof.

Clinical indicators used to evaluate the patients' cardiopulmonary status were arterial blood gas values, systemic blood pressure, body temperature, heart rate, respiratory frequency (f), tidal volume (V_T), and peak inspiratory flow (PIF). During FB, the f, V_T, and PIF were measured through the 7200a ventilator's patient data section. During the TP mode, f and V_T were measured with a Wright respirometer via a one-way valve. In order to calculate mean inspiratory flow (which, during TP trials, was the closest obtainable approximation of PIF), we employed the following formula:

$$\text{PIF (L/min)} = \frac{V_T \text{ (mL)} \times 60 \text{ (s/mn)}}{t_i \text{ (s)}},$$

where V_T and t_i (inspiratory time) are the average of three consecutive measurements.

Weaning from mechanical ventilation was accomplished by progressively decreasing the set rate in the SIMV mode on the ventilator, as well as progressively decreasing the pressure-support (PS) level, until the patient was on an SIMV rate of 6 breaths/min and a PS level of 5 cm H_2O. Then the patient was switched to the CPAP mode without PS. Patients who required PEEP were weaned down to 5 cm H_2O or less and an F_{IO_2} of 0.40.

The patients had to meet the following minimal criteria prior to being placed on FB or TP: normal mental status, intact gag reflex, stable vital signs, and acceptable arterial blood gas. A patient who met those criteria and was on CPAP of 5 cm H_2O or less and an F_{IO_2} of 0.40 was then placed on FB. At first the base flow was set at 10 L/min; then it was set at 20 L/min. The flow sensitivity was set at 3 L/min for both base flows. Measurements and data were collected after at least 20 minutes in the FB mode.

In the final step before extubation, each patient was placed on a valveless aerosol system consisting of a cold, all-purpose nebulizer, a 72-inch corrugated large-bore tubing, and a T-piece with a 6-inch reservoir tube. The nebulizer was set for 40% oxygen, and the flowmeter was set at 6 L/min, giving a total gas flow from the nebulizer of 24 L/min, thus establishing a base flow similar to the 20 L/min base flow in the FB mode and maximal peak inspiratory flows close to those desired prior to extubation. Again, all measurements and data were collected after at least 20 minutes on the TP system.

The patients were then re-evaluated for extubation, using institutional protocol, and once extubated they were given 40% oxygen via an aerosol mask.

Statistical Analysis

All variables were subjected to a three-level, one-way analysis of variance, with each technique (FB at 10 L/min, FB at 20 L/min, and TP) treated as a within-subjects factor.[6] $p \leq 0.05$ was accepted as statistically significant. The null hypothesis was that there was no statistically significant difference between measured variables in the various modes. Analysis was performed with SPSS/X software (SPSS Inc, Chicago IL).

Cost

Cost was evaluated by use of relative value units (RVU) for human resources, plus costs of added

material based on hospital contract costs. The cost of equipment and supplies already attached to the patient at the time of initiation of FB was not considered because the patient was already on the ventilator with its requisite tubing. The RVU assigned to initiating FB was 0.17 (ventilator adjustment—adult); for initiating TP it was 0.50 (weaning).[7] We used an hourly labor cost of $15.00, which was the average of departmental professional salaries that ranged from $12.52 to $16.65/hour without shift differentials or other adjustments.

Results

Weaning Data

Table 1 shows the weaning data for FB at 20 L/min, FB at 10 L/min, and the TP mode. Statistical analysis revealed a significant difference in P_{aCO_2} among the three modes, $F(2, 44) = 5.03$ ($p = 0.01$). Post-hoc comparisons showed that this effect resulted primarily from the difference between the P_{aCO_2} with FB at 20 L/min (mean 38 torr) and the P_{aCO_2} with the TP mode (mean 40 torr), $t(22) = 2.68$ ($p = 0.014$). The ANOVAs conducted on all other variables failed to reveal any significant effects. Correlations among all variables showed that no subject variables or conditional variables were associated with treatment or with any dependent measures to justify analyses of covariance.

Cost

The costs of equipment and supplies were $3.62 for a prefilled nebulizer, $0.63 for a 72-inch corrugated tube, and $0.15 for a T-piece. Table 2 displays these costs and shows that total costs were $2.55 each for FB at 20 L/min and 10 L/min, and $11.90 for TP.

Discussion

Our data reflect the clinical equivalence of the use of FB and TP in postsurgical mechanically ventilated patients prior to extubation (Table 1). The statistically significant difference between P_{aCO_2} during FB at 20 L/min (38 torr) and during TP

Table 1. Mean Values during Flow-By and T-Piece Ventilation

Variable	FB-20	FB-10	TP	F	p	SE
pH	7.40	7.41	7.41	0.43	0.65	0.01
P_{aO_2} (torr)	131	124	133	0.91	0.41	3.23
P_{aCO_2} (torr)	38*	39*†	40†	5.03	0.01	0.38
BP, systolic (torr)	146	151	150	1.10	0.34	1.92
BP, diastolic (torr)	67	70	71	0.88	0.42	1.41
S_{aO_2} (%)	98	98	98	0.70	0.50	0.30
Temperature (°F)	99	100	100	2.42	0.10	0.10
Pulse (beats/min)	93	92	95	1.22	0.30	1.40
Respiration (breaths/min)	22	22	23	2.01	0.17	0.31
Tidal volume (L)	0.44	0.42	0.43	0.38	0.68	0.01
PIF (L/min)	27	26	26	0.12	0.89	0.82

FB-20 = flow-by mode at 20 L/min; FB-10 = flow-by mode at 10 L/min; PIF = peak inspiratory flow; TP = T-piece mode.

*† Means not sharing a common superscript were significantly different as determined by post-hoc comparisons ($p = 0.05$). Differences were not clinically important.

(40 torr) is of questionable clinical importance because the difference in minute volume between these two values was only 0.3 L/min at a minute volume of 5.0 L/min.

Table 2. Costs of Setting Up Flow-By and T-Piece Modes

Variable	FB-20*	FB-10	TP
RVU (h)†	0.17	0.17	0.50
Labor Cost	$2.55	$2.55	$ 7.50
Equipment & Supplies Cost	0.00	0.00	4.40
Total Cost	$2.55	$2.55	$11.90

*FB-20 = flow-by mode at 20 L/min; FB-10 = flow-by mode at 10 L/min; TP = T-piece mode.
†RVU = relative value units; labor cost = (RVU) ($15.00).

Based on our RVU and hourly labor cost data, we found that instituting TP costs $9.35 more than instituting FB at either 10 or 20 L/min. This difference will change depending on RVUs, policies/procedures, and contract costs for different medical facilities. Consideration must also be given to the one-time cost of adding the flow-by option to the Puritan-Bennett 7200a ventilator, which is approximately $1,000. In the case of patients who otherwise would require prolonged weaning with frequent T-piece trials (eg, patients with COPD), the addition of flow-by may be cost-effective over time. Because an aerosol setup will be placed on most extubated patients—whether they are weaned by FB or TP—the cost differential for equipment alone may actually be less if weaning attempts are excluded. In addition, our study focused only on the cost during the pre-extubation period. If patients are placed on prolonged FB ventilation (eg, over several days) instead of on TP ventilation, the daily cost of ventilator usage has to be factored in.

In our center, we extubated 800 patients over the past year. If FB had been used instead of TP for all these patients, we would have saved about $7,480 as a result of decreased use of disposable supplies and decreased labor in facilitating the weaning process. In practice, not all patients require TP or FB trials prior to extubation, but in an era of limited resources, any potential savings would be welcome.

Conclusions

The major advantages of using the FB mode of the Puritan-Bennett 7200a ventilator are the internal fail-safe mechanisms and the easily accessible patient-monitoring system, which are essential if a patient becomes apneic or hypoventilates—both distinct possibilities, especially in a postanesthesia care unit, where narcotics are frequently used. In addition, weaning parameters can be obtained directly from the ventilator, thus eliminating the extra time needed to perform spirometry if a TP trial is used.

In summary, although our comparison of T-piece and flow-by ventilation prior to extubation in SICU patients found both modes to be clinically equivalent, the alarm and monitoring capabilities of the FB system are essential aids in a variety of clinical environments. Cost savings with the FB system may be realizable in prolonged weaning situations, but this requires further study.

ACKNOWLEDGMENTS

The authors thank Gary B Robert RRT for his assistance with the study, Andrew Manion MA BA for statistical consultation, and Lisa Fernandez and Phil Alkana RRT for their assistance in the preparation of this article.

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A New System for Understanding Mechanical Ventilators

Robert L Chatburn RRT

Come, let us go down and there confuse their language, that they may not understand one another's speech.

Genesis 11:7

Introduction

In many ways, mechanical ventilators have reached a level of sophistication comparable to personal computers. However, although ventilators sustain life whereas most personal computers are relegated to far less critical tasks, the average clinician probably understands less about ventilator design and operation than the average computer user understands about hard- and software. For example, consider the technical information supplied with personal computers. Most leading brands have well-organized and user-friendly operator's manuals. After-market textbooks and magazines are devoted to every aspect of a device's application, with detailed feature-by-feature comparisons among computer brands. Users' groups, bulletin boards, and newsletters abound.

In contrast, such detailed technical information does not exist for even the most popular mechanical ventilators. I have observed that compared with their computer counterparts, ventilator operator's manuals are poorly organized, difficult to use (some even lack indexes), and badly produced (one well-established manufacturer distributes a manual with crude, hand-drawn illustrations!). Textbooks offer vaguely defined and contradictory terminology based on outdated classification systems—systems that lack the flexibility and theoretical engineering basis required to match today's technologic advances. Outside of an occasional newsletter or trade journal produced by manufacturers, no additional information exists. Scientific articles about a given device seldom address the daily operational concerns of clinicians.

Our profession's approach to mechanical ventilators is hampered by the lack of an adequate classification system. Authors[1-7] have strung together loose collections of unrelated facts rather than developing a system of logical, related ideas linked to a few basic engineering principles. Their classification schemes have been founded on descriptions of mechanical archetypes (such as constant vs nonconstant pressure generator or constant vs nonconstant flow generator). I believe the problem with this approach to be that it quickly becomes outdated and restrictive as technology evolves and archetypes become irrelevant or obsolete. Thus, the concepts of ventilator design presented in most textbooks are too narrowly defined, if they are explicitly defined at all. As a consequence, readers miss the big picture and the understanding necessary to cope with today's science. For example, many clinicians assume that infant ventilators are somehow fundamentally different from adult ventilators and thus fail to see how they might be used interchangeably. Less expensive ventilators are seen as somehow inferior to more expensive models because they do not have the same dials or as many push buttons on their front panels. Manufacturers compound the problem by inventing their own definitions as new products develop, and they naturally tend to emphasize the uniqueness of their products' features as distinct from those of other manufacturers.

As a result of all this, clinicians are being forced to memorize a growing list of arbitrary device at-

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tributes and jargon. For example, at a recent national meeting, a prototype ventilator was shown that had no fewer than seven modes of ventilation, some designated by compound acronyms. One mode was called Pressure Regulated Volume Control. A quick check of the dictionary shows that to regulate means to control. Thus, the name is confusing because it is internally inconsistent, implying that both pressure and volume are controlled. I will show that such dual control is impossible from the standpoint of mathematics, pulmonary mechanics, or engineering. The name ignores the fact that most clinicians correctly view pressure and volume control as two distinctly different modes of ventilation. Even the manufacturer in question has used the terms volume control and pressure control as different modes on a selector switch. Pity the poor sales representative who must explain such inconsistencies to potential customers. What about the customers who have to explain it to their colleagues? And, what of educators and their bewildered students? They have all been reduced to hand-waving in a feeble attempt to get people to understand what they mean rather than what they say.

New modes of ventilation spring up like mushrooms, are named arbitrarily, and are tacked on to the ventilator's mode-selector switch like so many plastic magnets on a refrigerator door.

Even worse, a certain mode may be defined differently by different manufacturers. For example, a breath in the pressure-support mode may be terminated when the inspiratory flow decays to 25% of peak flow or when it reaches 5 L/min depending on the manufacturer. One manufacturer defines mandatory minute ventilation in terms of automatic control of ventilatory frequency, whereas another defines it in terms of automatic control of pressure-support level. Two manufacturers may define a mode the same way but call it by two different names. And without fail, each of these new modes is described by paragraphs of "when the patient does this, the ventilator does this, but when this happens, that happens . . .." Such descriptions are hard to follow, hard to memorize, and even harder to explain to someone else.

Ten years ago, it was easy to remember assist/control, intermittent mandatory ventilation (IMV), and even synchronized intermittent mandatory ventilation (SIMV). Two or three modes on one ventilator were manageable. Today, we face two possibilities—either we go on memorizing an endless list of manufacturer-specific features, or we find a new way to think about ventilators. Again, there is an analogy in the computer world. Those who use computers employing MS-DOS operating systems (IBM and clones) must memorize a different list of cryptic, abbreviated, and unrelated disk-operating commands for each program, to control their computers. As a result, the average IBM user is familiar with only two or three programs—perhaps a word processing and a spreadsheet program. On the other hand, Apple Macintosh users are provided with familiar plain-English commands and simple icons that are obvious and for the most part consistent from one program to the next. As a result, the average Macintosh user is familiar with more programs, and the learning time for new programs is greatly reduced. (Many Macintosh operators do not even read the program manuals because the commands are easily recognized.)

The purpose of this article is to propose a new way of thinking about mechanical ventilators. The system simplifies ventilator identification by focusing on similarities rather than differences. The classification scheme introduced here is based on a simple mathematical model of respiratory-system mechanics rather than mechanical archetypes. Because the basic concepts of respiratory-system mechanics do not change with time, this scheme is immune to changes in ventilator technology. Indeed, you will see that almost any conceivable increase in technologic complexity can be described without increasing confusion, by applying a small set of definitions and concepts as included in the Glossary at the end of the text. In fact, any mode of ventilation can be described completely by simply specifying the variables that start, sustain, and end inspiration for both mandatory and spontaneous breaths. This approach unmasks similarities and differences hidden by the jargon used to name ventilatory modes. For example, when viewed in this manner, the so-called airway pressure-release mode of ventilation is seen to be a time-triggered, pressure-limited, and time-cycled mode, with spontaneous breaths passively supported by a continuous

flow of gas through the patient circuit. This is *identical* to the technique applied universally by all modern infant ventilators. The emperor has no clothes! Airway pressure release is not a new mode but simply unnecessary jargon for pressure-controlled ventilation. It is true that the peak- and baseline-pressure levels may be somewhat different from usual, but the principles of ventilation and ventilator control are not.

Finally, the proposed system provides a practical and consistent theoretical framework for teaching and learning ventilator operation that many educators have found lacking in contemporary textbooks. The Appendix provides a practical approach to testing performance.

Basic Concepts

A ventilator is simply a machine—a system of related elements designed to alter, transmit, and direct applied energy in a predetermined manner to perform useful work.[8] We put energy into the ventilator in the form of electricity (energy = volts × amperes × time) or compressed gas (energy = pressure × volume). That energy is transmitted or transformed (by the ventilator's drive mechanism) in a predetermined manner (by the control circuit) to augment or replace the patient's muscles in performing the work of breathing (the desired output). Therefore, to understand mechanical ventilators, we must first understand their basic functions.

I. Power Input

II. Power Transmission or Conversion

III. Control

IV. Output (pressure, volume, and flow waveforms)

This outline can be expanded to add as much detail about a given ventilator as desired.

I. Input
  A. Pneumatic
  B. Electric
    1. Alternating current, or AC
    2. Direct current, or DC (battery)

II. Power Conversion and Transmission (drive mechanism)
  A. External Compressor
  B. Internal Compressor
    1. Motor and linkage
      a. Compressed gas, direct
      b. Electric motor—rotating crank and piston rod
      c. Electric motor—rack and pinion
      d. Electric motor—direct
  C. Output Control Valves
    1. Pneumatic diaphragm
    2. Pneumatic poppit valve
    3. Electromagnetic poppit valve
    4. Electromagnetic proportional valve

III. Control
  A. Control Circuit
    1. Mechanical
    2. Pneumatic
    3. Fluidic
    4. Electric
    5. Electronic
  B. Control Variables and Waveforms
    1. Pressure
    2. Volume
    3. Flow
    4. Time
  C. Phase Variables
    1. Trigger
    2. Limit
    3. Cycle
    4. Baseline
  D. Conditional Variables

IV. Output
  A. Pressure Waveforms
    1. Rectangular
    2. Exponential
    3. Sinusoidal
    4. Oscillating
  B. Volume Waveforms
    1. Ramp
    2. Sinusoidal
  C. Flow Waveforms
    1. Rectangular
    2. Ramp
      a. Ascending
      b. Descending
    3. Sinusoidal

D. Effects of the Patient Circuit

V. Alarm Systems
- A. Input Power Alarms
  - 1. Loss of electric power
  - 2. Loss of pneumatic power
- B. Control Circuit Alarms
  - 1. General systems failure (ventilator inoperative)
  - 2. Incompatible ventilator settings
  - 3. Inverse inspiratory-to-expiratory ratio
- C. Output Alarms
  - 1. Pressure
    - a. High and low peak airway pressure
    - b. High and low mean airway pressure
    - c. High and low baseline pressure (PEEP) or CPAP)
    - d. Failure of airway pressure to return to baseline within a specified period
  - 2. Volume
  - 3. Flow
  - 4. Time
    - a. High or low ventilatory frequency
    - b. High or low inspiratory time
    - c. Long or short expiratory time (long expiratory time = apnea)
  - 5. Inspired gas
    - a. High or low inspired gas temperature
    - b. High or low F_{IO_2}
  - 6. Expired gas
    - a. Exhaled carbon dioxide tension
    - b. Exhaled oxygen tension

A discussion of input power sources (I) and power conversion and transmission (II) is beyond the scope of the article. However, I explore, in detail, control schemes and ventilator output (ie, pressure, volume, and flow) and apply the classification system to alarms. Finally, I suggest some procedures for evaluating ventilator performance.

Classifying Mechanical Ventilators

A good ventilator classification scheme should describe how a given ventilator works in general terms but have enough detail to allow the one particular ventilator to be distinguished from others. A good scheme facilitates description by focusing on key attributes or characteristics in a logical and consistent manner. A clear description allows us to quickly assess new facts in relation to our previous knowledge. Learning the operation of a new device or describing it to others then becomes much easier. If we understand how the ventilator operates, we can anticipate appropriate ventilator management strategies for particular clinical situations.

In this section I review the basic concepts of control theory and how they apply to mechanical ventilators. But, first, we must review the even more fundamental ideas about what we are controlling, namely, the motion of the respiratory system itself.

To understand how a machine can be controlled to replace or supplement the natural function of breathing, we need to understand something about the mechanics of breathing. The study of mechanics deals with forces, displacement, and the rate of change of displacement. In physiology, force is measured as pressure (pressure = force/area), displacement is measured as volume (volume = area × displacement), and the relevant rate of change is measured as flow (eg, average flow = Δvolume ÷ Δtime; instantaneous flow = dv/dt, the derivative of volume with respect to time). We are interested in the *pressure* necessary to cause a *flow* of gas to enter the airway and increase the *volume* of the lungs.

Pressure, volume, and flow all change with time over the course of one inspiration and expiration. The relation among them is described by a mathematical model known as the equation of motion for the respiratory system.[9] (The equation is a linear differential equation with constant coefficients that uses calculus to obtain any one of the three variables as a function of the other variables and time. Volume is the integral of flow with respect to time, and flow is the derivative of volume with respect to time.) In simplified form the equation is

$$muscle\ pressure + ventilator\ pressure = \frac{volume}{compliance} + (resistance)(flow), \quad [1]$$

where muscle pressure is the 'imaginary' transrespiratory pressure (ie, airway pressure – body surface pressure) generated by the ventilatory muscles to expand the thoracic cage and lungs.

Muscle pressure is said to be imaginary because it is not directly measurable. Ventilator pressure is the transrespiratory pressure generated by the ven-

tilator during inspiration. The combined muscle and ventilator pressure causes volume and flow to be delivered to the patient. Pressure, volume, and flow change with time and hence are variables. Compliance and resistance are assumed to remain constant and are called parameters; their combined effect constitutes the load experienced by the ventilator and ventilatory muscles.

It is important to remember that pressure, volume, and flow are all measured relative to their baseline values (ie, their values at end-expiration). This means that the pressure that causes inspiration is measured as the change in airway pressure above positive end-expiratory pressure (PEEP). (Airway pressure as reflected by the ventilator manometer at the patient's airway opening is actually transrespiratory pressure because the ventilator manometer reads gauge pressure, pressure relative to atmospheric or body surface pressure.) This is the reason, for example, that pressure-support levels are measured relative to PEEP. Thinking of ventilator pressure simply as airway pressure (ie, pressure measured at one point in space, the airway) limits understanding of the mechanics involved in breathing (eg, during 'negative-pressure' ventilation, airway pressure does not change during inspiration, but transrespiratory pressure changes just as in positive-pressure ventilation). Volume is measured as the change in lung volume above functional residual capacity (FRC), and the change in lung volume during the inspiratory period is defined as the tidal volume (V_T). Flow is measured relative to its end-expiratory value (usually zero). These concepts are illustrated in Figure 1.

Note that if the patient's ventilatory muscles are not functioning, muscle pressure = 0, and the ventilator must generate all of the pressure required to deliver the tidal volume and inspiratory flow. On the other hand, a ventilator is not needed for normal, spontaneous breathing (ie, ventilator pressure = 0). Between these two extremes, an infinite number of combinations of muscle pressure (ie, patient effort) and ventilator support is theoretically possible. These combinations suggest a great many 'modes' of ventilation for partial ventilatory support.

Perhaps, the most important insight provided by the equation of motion is that any ventilator can directly control only one variable at a time: pressure, volume, or flow. Therefore, we can think of a ventilator simply as a machine that controls either the airway pressure waveform, the inspired volume waveform, or the inspiratory flow waveform (a waveform is a plot of a variable's magnitude on the vertical axis vs time on the horizontal axis). Thus, pressure, volume, and flow are referred to in this context as control variables. Time is a variable that is implicit in the equation of motion. In some cases provided later, time is viewed as a control variable.

The ventilator's control scheme may be sophisticated, with the entire shape of the waveform predetermined during inspiration. Many third-generation ventilators can do this and even offer a choice of waveforms. On the other hand, the control scheme may be quite simple and with only one waveform characteristic, such as the peak or mean value, controlled during inspiration. A common example is the way that an infant ventilator controls pressure simply by limiting the peak inspiratory value.

Having determined *what* is controlled, we can now explore *how* it is controlled. For our purpose, a system is a collection of elements that interact according to some particular process or function. A model is a simplified version of a real-world system used to help us understand the relationships among system elements (eg, the equation of motion is a model of the respiratory system). We need to understand the relation between the input and the output of the system (ie, we need to create a model). This understanding may then help us to control the system behavior.

A system can be controlled in two different ways to achieve the desired output.[10]

1. Select an input and wait for an output with no interference during the waiting period.
2. Select an input, observe the trend in the output, and modify the input accordingly to attain, as nearly as possible the desired output.

For example, when a helmsman steers a boat towards the dock, he may do it in one of two ways:

1. by pointing the boat in the direction of the dock and retiring to his cabin or

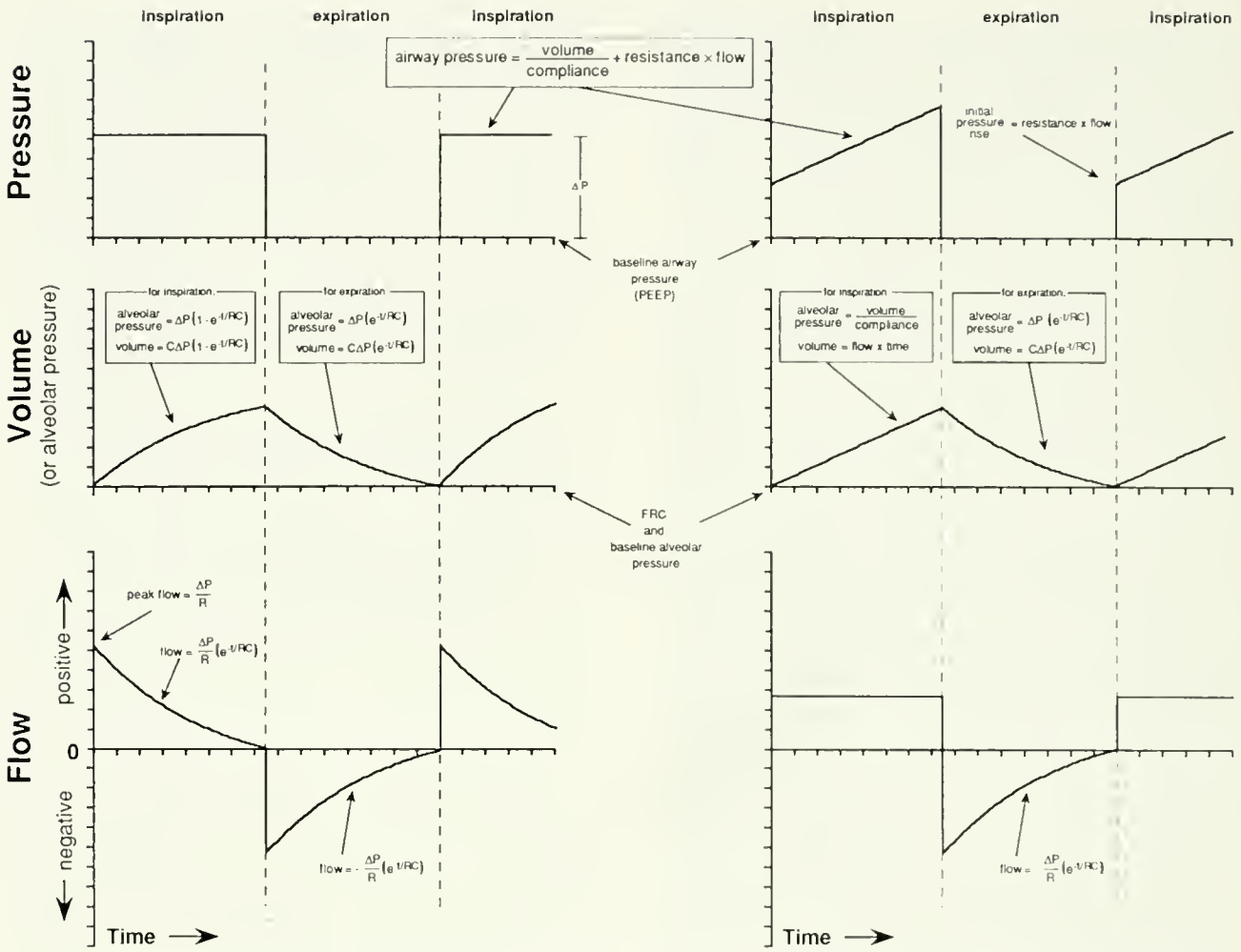

Fig. 1. Typical pressure, volume, and flow waveforms for pressure-controlled (rectangular pressure waveform) and volume-controlled (rectangular flow waveform) ventilation. Pressure, volume, and flow are shown as functions of time in accordance with the equation of motion (where muscle pressure = 0). Note that all variables are measured relative to their baseline, or end-expiratory values. ΔP = change in airway pressure; R = resistance; C = compliance; t = time; e = base of natural logarithm (≈ 2.72), FRC = functional residual capacity.

2. by continuously adjusting the boat's heading based on observation of the relative positions of the dock and the moving boat.

In this example, the system is the boat (motor, propeller, and steering mechanism); the input is the position of the boat's steering wheel; and the output is the direction of the boat's motion. With both approaches to control, a change in the input causes a change in the output, but in the first case there is no flow of information from the output to generate a new input to 'close the loop.' Hence, this type of control scheme is called open-loop control. In the second case, the helmsman uses information about the output to modify the input, which in turn improves the output. This control scheme is called closed-loop, feedback, or servo control. Block dia-grams in Figure 2 illustrate open- and closed-loop control systems.

Note that to achieve closed-loop control, the output must be measured and compared to a reference value. In the boat example, a human (the helmsman) measures and compares functions; but, in a ventilator, a transducer and electronic circuitry are necessary to perform automatic closed-loop control. Closed-loop control provides the advantage of a more consistent output in the face of unanticipated disturbances. In the boat example, dis-

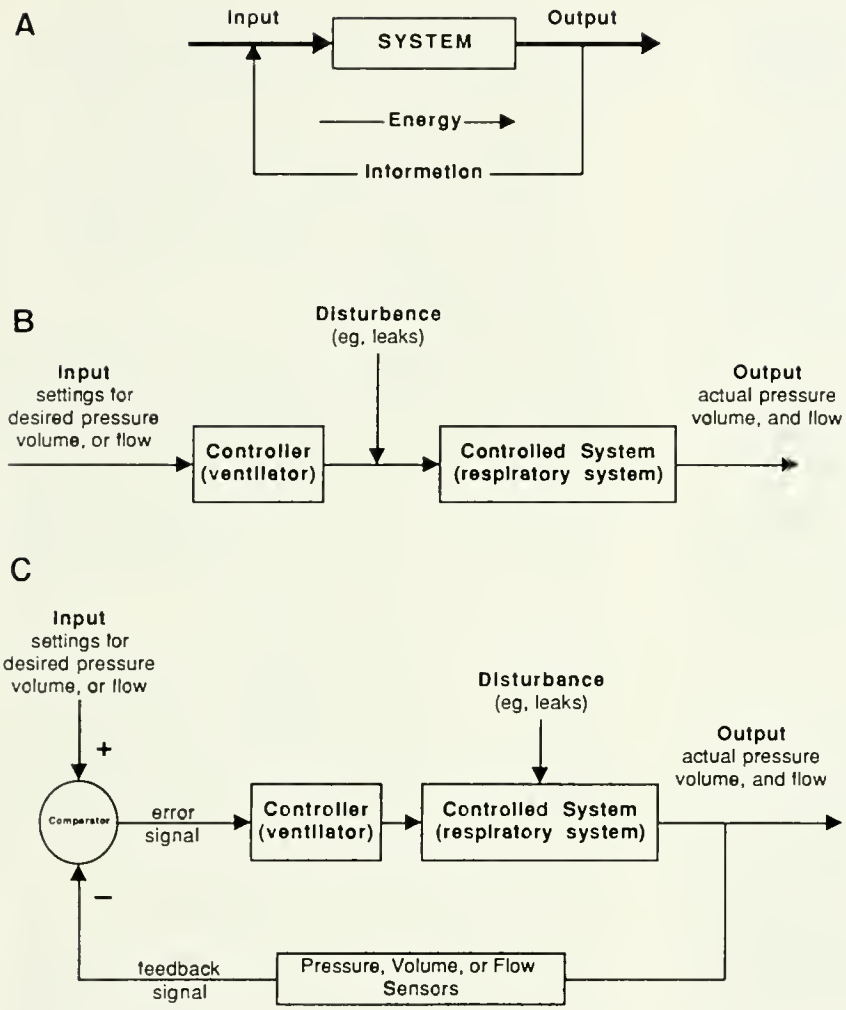

Fig. 2. A—A system with one input and one output. Energy flows from input to output, and information flows from output to input. B—Example of open-loop control. C—Closed-loop, feedback, or servo control.

turbances that affect the direction of the boat might include wind and water currents. In the case of ventilators, disturbances that might affect the delivery of pressure, volume, and flow include pooled condensation or leaks in the patient circuit, endotracheal-tube obstructions, and changes in respiratory-system resistance and compliance.

Ventilator designs incorporate both open- and closed-loop control. For example, both open- and closed-loop pressure control have been used in the design of infant ventilators. The Newport Breeze uses open-loop control to generate the inspiratory pressure waveform in its pressure-control mode. The operator sets a flowrate and inspiratory pressure limit. The pressure-limit setting determines the pressure on one side of a pneumatic diaphragm in the exhalation manifold. Inspiratory pressure rises as the lungs and patient circuit fill with gas. Pressure rise is halted when pressure in the system reaches the pressure applied to the pneumatic diaphragm. At this point, ventilator gas spills out of the circuit into the atmosphere. This is an example of open-loop control because inspiratory pressure is not measured for the purpose of controlling the flow of gas from the ventilator. Thus, if a leak should occur in the patient circuit, the peak inspiratory pressure may not be met, and the ventilator would not be able to alter its flow output to correct for the problem.

In contrast, the Infrasonics Infant Star uses inspiratory pressure as a feedback signal to vary the inspiratory flowrate in order to maintain a constant inspiratory pressure when set in the IMV mode. Because of its ability to use closed-loop control, the Infant Star can maintain a more consistent peak inspiratory pressure than the Breeze in the face of patient-circuit or respiratory-system changes. Most third-generation adult mechanical ventilators use

closed-loop control of pressure and flow waveforms.

A. Control Circuit

The control circuit is the subsystem responsible for controlling the drive mechanism and/or the output control valve. A ventilator may have more than one control circuit, which may be of several types.

1. Mechanical. Mechanical control circuits use levers, pulleys, and cams. These types of circuits were used in the early manually operated ventilators.[11]

2. Pneumatic. Pneumatic control circuits use gas pressure to operate diaphragms, jet entrainment devices, venturis, and pistons. The original Bird and the Bennett PR-series ventilators used pneumatic control.

3. Fluidic. Fluidic circuits are analogs of electronic logic circuits.[12] They use minute gas flows to generate signals that operate timing systems and pressure switches. This makes them immune to failure from electromagnetic interference (such as from magnetic resonance imaging equipment). Fluidic circuits can be constructed with discrete components like comparators and flip-flops or they can be combined in integrated circuits, analogous to electronic integrated circuits. Examples of ventilators using fluidic-logic control circuits are the Sechrist IV-100B and the Bio-Med MVP-10.

Fluidic devices operate on a principle known as the Coanda effect. This effect is observed when a free-flowing stream of gas passes near a rigid wall. The rapidly flowing gas tends to entrain molecules of the surrounding stationary gas.[13] As molecules are removed from the confined area between the stream and the wall, the ambient pressure drops compared to the other side of the stream (where molecules from the atmosphere rush in to replace those dragged into the stream). Thus, the resultant pressure differential tends to force the stream to flow alongside of or 'attach itself' to the wall.

The effect was named after Dr Henri Coanda, a Rumanian aeronautical engineer, who discovered it on his first and last test flight of a jet engine aircraft in 1910.[14] According to Coanda,

> Apparently I had given it too much fuel—when I looked over the side, I saw flames shooting out, and that should not be. Not with my wooden wings full of petrol. I ducked back inside to adjust matters. A moment later things felt very differently. I looked outside again to find myself many feet in the air. Straight ahead of me was the Paris wall. I didn't know what to do. I pulled on the control wheels, the machine went up on one wing and I was thrown out. The machine crashed right at the foot of the wall.

Later, Coanda discovered that the mica deflection plates he had used to direct the exhaust away from the plane had actually turned the flames toward it.

4. Electric. Electric control circuits use only simple switches to control ventilator operation. One example of a completely electrically controlled ventilator is the Emerson Iron Lung.

5. Electronic. Electronic control circuits use devices such as resistors, capacitors, diodes, and transistors and combinations of these components in integrated-circuits. Integrated circuits can range in complexity from simple logic gates and operational amplifiers to microprocessors.

B. Control Variables and Waveforms

All ventilator control schemes are designed to accomplish one goal: to support some fraction of the patient's minute volume. Figure 3 is a simplified influence diagram[15,16] that illustrates the important variables for ventilators that are either volume- or flow-controllers (adapted from Perry[17]). The equations relating these variables are explained extensively elsewhere.[18] Figure 4 is the influence diagram for ventilators that are pressure-controllers (adapted from Chatburn and Lough[19]).

A limited number of waveforms are commonly used by current ventilators for each control variable and can be idealized and grouped into four basic categories: rectangular, exponential, ramp, and sinusoidal (Fig. 5). The meaning of these terms should be obvious from inspection of the waveforms shown in Figure 5 and will be explained in more detail in the section on Output. (Note that a rectangular volume waveform is theoretically impossible because volume cannot change instantaneously from zero to some preset value as pressure

and flow can.) Thus, a ventilator can be classified as either a pressure-, volume-, or flow-controller, and may be further characterized by the types of waveforms it can generate. In some cases, it is logical to classify a ventilator as a time-controller (ie, it controls only inspiratory and expiratory time).

Any given ventilator may control more than one variable (at different times), depending upon how it is used. For example, the Bennett 7200a ventilator can mix flow-controlled breaths with pressure-controlled breaths in the SIMV + Pressure Support mode. The great flexibility of today's ventilators is achieved at the expense of simplicity. Thus, when evaluating ventilator performance, it is important to have simple and unambiguous criteria for deciding which control variables are in effect. The criteria both necessary and sufficient to differentiate the four possible control variables are shown in Figure 6. These criteria are based on the effect of the load on the ventilator's output.

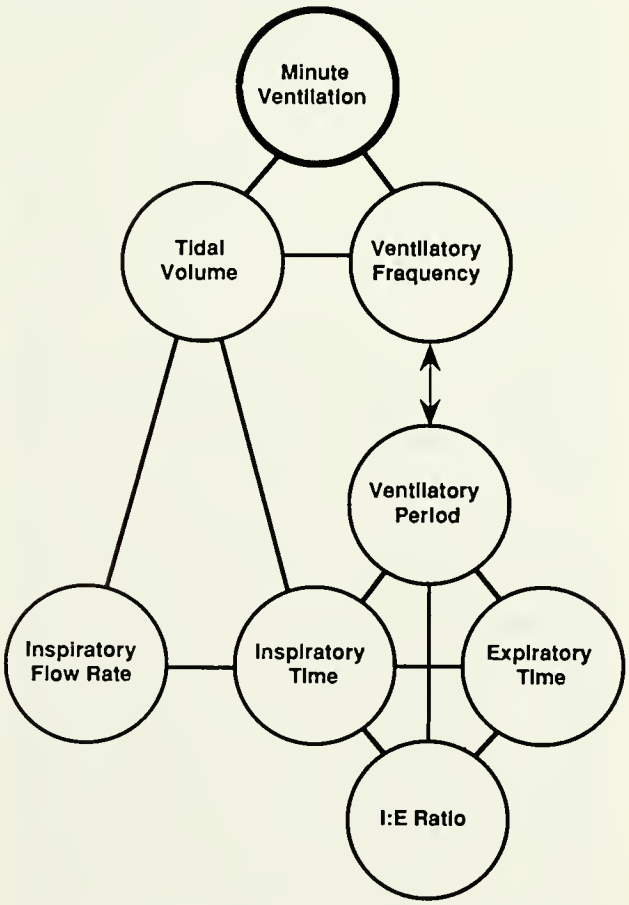

Fig. 3. Influence diagram for volume-controlled ventilation. Variables are connected by straight lines indicating that if any two are known, the third can be calculated using standard equations.[18] The double arrow line indicates that ventilatory period is the reciprocal of ventilatory frequency.

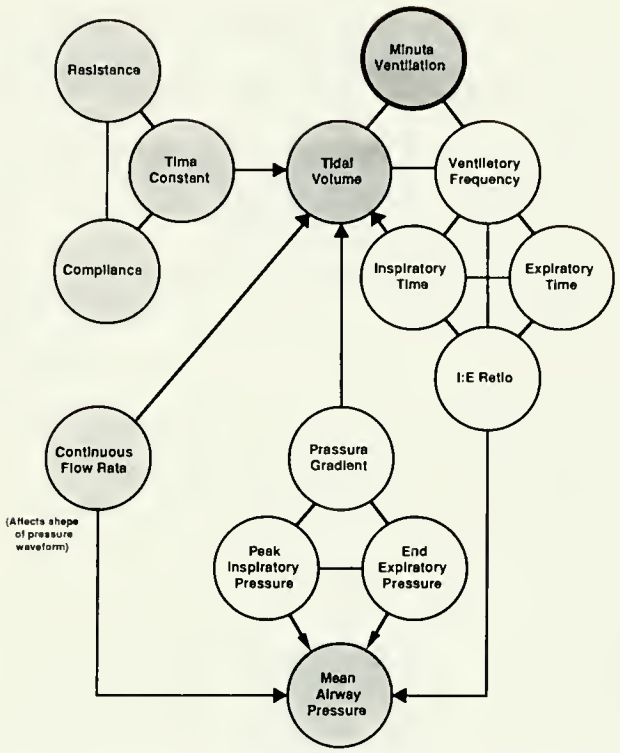

Fig. 4. Influence diagram for pressure-controlled ventilation. Variables are connected by straight lines indicating that if any two are known, the third can be calculated using standard equations.[18] Arrows suggest that relationships are either more complex or less predictable than others shown. Open circles represent variables that can be directly controlled by the ventilator, shaded circles are controlled indirectly.

The respiratory system, as described by the equation of motion, is analogous to an electrical circuit composed of a capacitor and resistor connected in series. A pressure-controller is analogous to a constant voltage source, whereas a flow- or volume-controller is analogous to a constant current source. Using this analogy, if the ventilator is a pressure-controller, a respiratory system with a high compliance and a low resistance appears as a large load to the ventilator and tends to deform the pressure waveform. On the other hand, if the ventilator is a volume- or flow-controller, a high re-

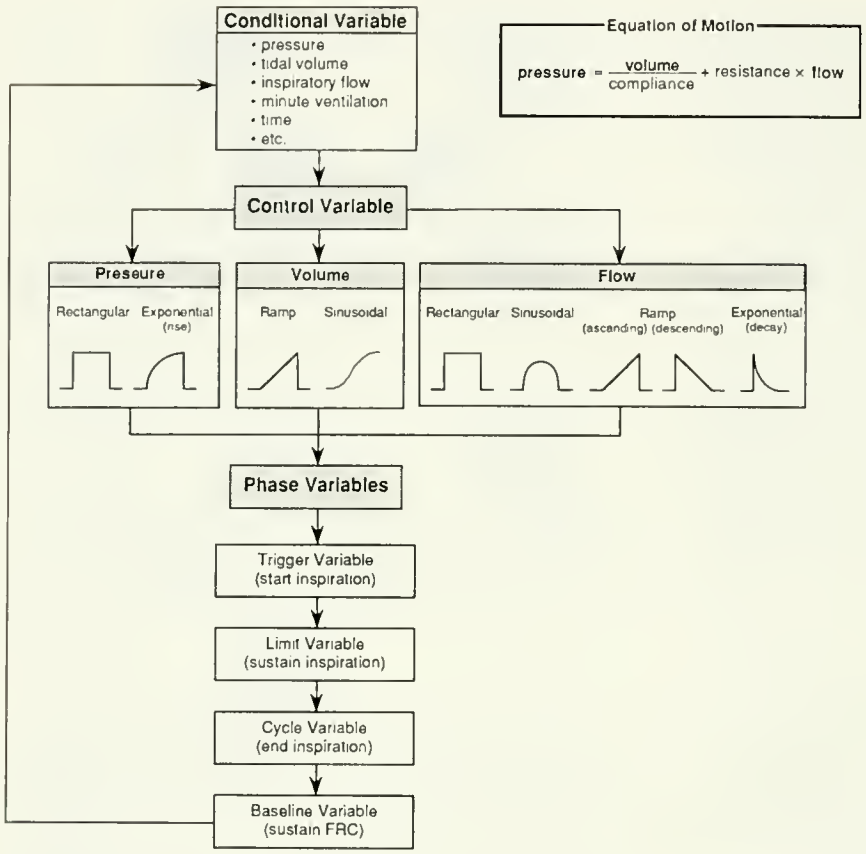

Fig. 5. Application of the equation of motion for the respiratory system. Common waveforms for each control variable are shown. Pressure, volume, flow, and time are also used as *phase variables* that determine the characteristics of each ventilatory cycle (eg, trigger sensitivity, peak inspiratory flowrate or pressure, inspiratory time, and baseline pressure). The flowchart depiction emphasizes that each breath may have a different set of control and phase variables depending on the mode of ventilation desired.

Table 1. Idealized Load Effects for Pressure- and Flow-Controllers

Ventilator Type	Respiratory Parameters*	Load Effect	Desired Waveform	Actual Waveform
Pressure-Controller	↑ C, ↓ R	large		
	↑ C, ↑ R	medium		
	↓ C, ↓ R	medium		
	↓ C, ↑ R	small		
Flow-Controller	↑ C, ↓ R	small		
	↑ C, ↑ R	medium		
	↓ C, ↓ R	medium		
	↓ C, ↑ R	large		

* ↑ = high; ↓ = low; C = compliance; R = resistance.

sistance and a low compliance appears as a large load to the ventilator, and the volume or flow waveform may be deformed. Some generalized loading effects are illustrated in Table 1.

The criteria in Figure 6 are based on the concepts embodied in the equation of motion (assuming that muscle pressure = 0) and the control theories developed above. An important conse-quence of these theories is that for a ventilator to use feedback to control a variable, it must first be able to sense the variable. This implies some type of transducer (eg, pressure gauge, volume displacement measuring device, or flow transducer). Note that a calibrated dial on the control panel of a ventilator does not necessarily imply that the set variable is being measured and controlled. Keeping these ideas in mind will help to distinguish between volume- and flow-controllers.

1. Pressure. If the control variable is pressure, the ventilator can control either the airway pressure

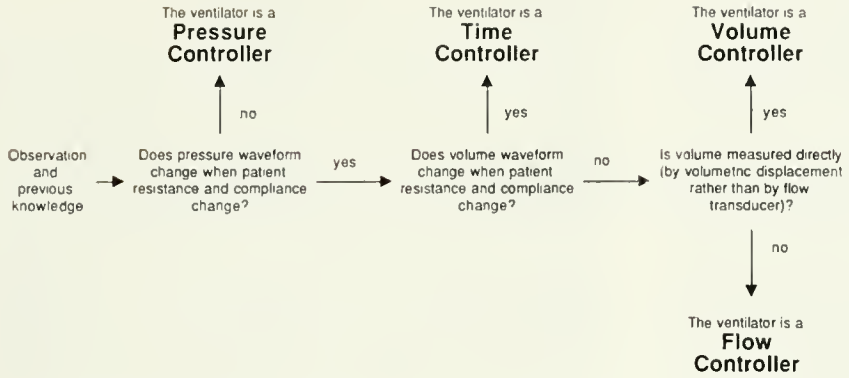
Observation and previous knowledge
Does pressure waveform change when patient resistance and compliance change?
no
The ventilator is a Pressure Controller
yes
Does volume waveform change when patient resistance and compliance change?
no
The ventilator is a Time Controller
yes
Is volume measured directly (by volumetric displacement rather than by flow transducer)?
no
The ventilator is a Volume Controller
The ventilator is a Flow Controller

Fig. 6. Criteria for determining the control variable during a ventilator-supported inspiration. Beginning with observations and previous knowledge of this ventilator, decisions are based on the effect of load on ventilator output.

(causing it to rise above body surface pressure for inspiration) or the pressure on the body surface (causing it to fall below airway opening pressure for inspiration). This is the basis for classifying a ventilator as either a positive- or negative-pressure type. (The terms positive-pressure and negative-pressure ventilator obscure the fact that both devices generate a positive transrespiratory pressure.) For example, the Newport Wave ventilator can be classified as a positive-pressure controller that generates a rectangular pressure waveform, and the Emerson Iron Lung as a negative-pressure controller that produces a quasi-sinusoidal pressure waveform.

The equation of motion tells us that if the ventilator is an ideal pressure-controller, then the left side of the equation (ie, ventilator pressure as a function of time) is determined by the ventilator settings and is unaffected by changes in parameter values on the right side (ie, compliance and resistance).

2. Volume. If the pressure waveform varies as the load changes, we then examine the volume waveform. However, the observation that the volume waveform remains unchanged is a necessary but not a sufficient condition to warrant the classification of volume-controller because the same holds true for a flow-controller. Once the volume waveform has been specified, the flow waveform is determined because they are functions of each other (ie, volume is the integral of flow, and flow is the derivative of volume). Therefore, if changes in compliance and resistance do not change the volume waveform, they will not affect the flow waveform and vice versa.

The distinguishing feature of a volume-controller is that it measures the volume it delivers. Delivered volume can be measured directly only by the volume change of the ventilator's compressor, such as the displacement of a piston or bellows.[20] Therefore, the only ventilators that qualify as volume-controllers are those with drive mechanisms that (1) maintain an approximately constant volume change in the face of a varying load; (2) employ a compressor such as a piston, bellows, or similar device; and (3) measure volume change as the displacement of the piston, bellows, or similar mechanism. Two examples are the Emerson 3MV and the Bennett MA-1. Although some ventilators like the Siemens Servo 900C, the Bennett 7200, the Bear 5, and the Hamilton Veolar display volume readings, they all actually measure flow and calculate volume. Thus, they are all flow-controllers unless they are operated in a pressure-control mode (eg, during pressure-support ventilation). An examination of a ventilator's schematic diagrams and operator's manual should provide the information necessary to decide whether volume or flow is being measured.

3. Flow. If the volume change (ie, tidal volume) remains consistent when compliance and resistance are varied, and if volume change is not directly measured, the ventilator is classified as a flow-controller. The simplest example of open-loop flow control in a ventilator consists of a pressure regulator and a flowmeter such as are found in infant ventilators. An infant ventilator becomes a flow-controller rather than a pressure-controller if the airway pressure does not reach the set pressure limit.[21] (However, the flowmeter is usually not back-pressure compensated and varies its output slightly in the face of a changing load.) In contrast, the Siemens Servo 900C measures flow and adjusts the

output-control valve (ie, the inspiratory scissors valve) accordingly. It can maintain a more consistent inspiratory flow waveform as the load changes.

4. Time. If both pressure and volume are affected substantially by changes in lung mechanics, then the only form of control is that of defining the ventilatory cycle or alternating between inspiration and expiration. Therefore, the only variables being controlled are the inspiratory and expiratory times. This situation arises in some forms of high-frequency ventilation in which even the designation of an inspiratory and expiratory phase becomes somewhat obscure.

C. Phase Variables

Once the control variables and the associated waveforms are identified, more detail can be obtained by examining the events that take place during a ventilatory cycle (that is, the period of time between the beginning of one breath and the beginning of the next). Mushin et al[14] proposed that this time span be divided into four phases: the change from expiration to inspiration, inspiration, the change from inspiration to expiration, and expiration. This convention is useful for examining how a ventilator starts, sustains, and stops an inspiration and what it does between inspirations. In each phase, a particular variable is measured and used to start, sustain, and end the phase. In this context, pressure, volume, flow, and time are referred to as phase variables[22] (Fig. 5).

1. Trigger. All ventilators measure one or more of the variables associated with the equation of motion (ie, pressure, volume, flow, or time). Inspiration begins when one of these variables reaches a preset value. Thus, the variable of interest is considered an initiating or trigger variable. The most common trigger variables are time (ie, the ventilator initiates a breath according to a set frequency, independent of the patient's spontaneous efforts) and pressure (ie, the ventilator senses the patient's inspiratory effort as a drop in baseline pressure and starts inspiration independent of the set frequency). Of course it may be possible to manually trigger inspiration.

It is feasible for a ventilator to measure volume and flow changes caused by the patient's inspiratory efforts and use them to trigger inspiration, but the technology is more complex and is not commonly used. Triggering on flow has been shown to be more sensitive than triggering on pressure (at least with conventional patient circuits), hence the patient has to do less work to receive a breath.[23] Currently, only the Bennett 7200a can be flow triggered and only the Dräger Babylog is volume triggered. The criteria for determining the trigger variables of a particular ventilator are shown in Figure 7.

The patient effort required to trigger inspiration is determined by the ventilator's sensitivity. Sensitivity is adjusted by changing the preset value of the trigger variable. For example, to make a pressure-triggered ventilator more sensitive, the trigger pressure is adjusted from 5 cm H_2O below to 1 cm H_2O below baseline pressure.

Some ventilatory modes permit the patient to breathe spontaneously between mandatory breaths. But an interesting dilemma ensues: What is the difference between a mandatory and a spontaneous breath? Again, this is a problem born of technologic evolution. Taber's definition of spontaneous is "occurring . . . without apparent cause; voluntary."[24] It is clear, for example, that a patient breathes spontaneously when not connected to a ventilator—the patient can control the frequency and size of breaths and will hypoventilate if apnea occurs. Originally, ventilators controlled every breath. The idea that there could be such a thing as a spontaneous breath during mechanical ventilation arose when continuous-flow IMV was introduced. Yet, it is obvious, in the context of IMV, that mandatory breaths are triggered, or caused, by the ventilator and spontaneous breaths are under voluntary control of the patient. SIMV was created as a refinement of IMV in which the ventilator responds to patient breathing efforts in one of two ways, depending upon when the effort occurs. A consideration of this mode complicates matters. If a patient inspiratory effort occurs at the 'right' time, inspiration will be pressure (or flow) triggered instead of time triggered by the ventilator. The right time is usually referred to as a window or per-

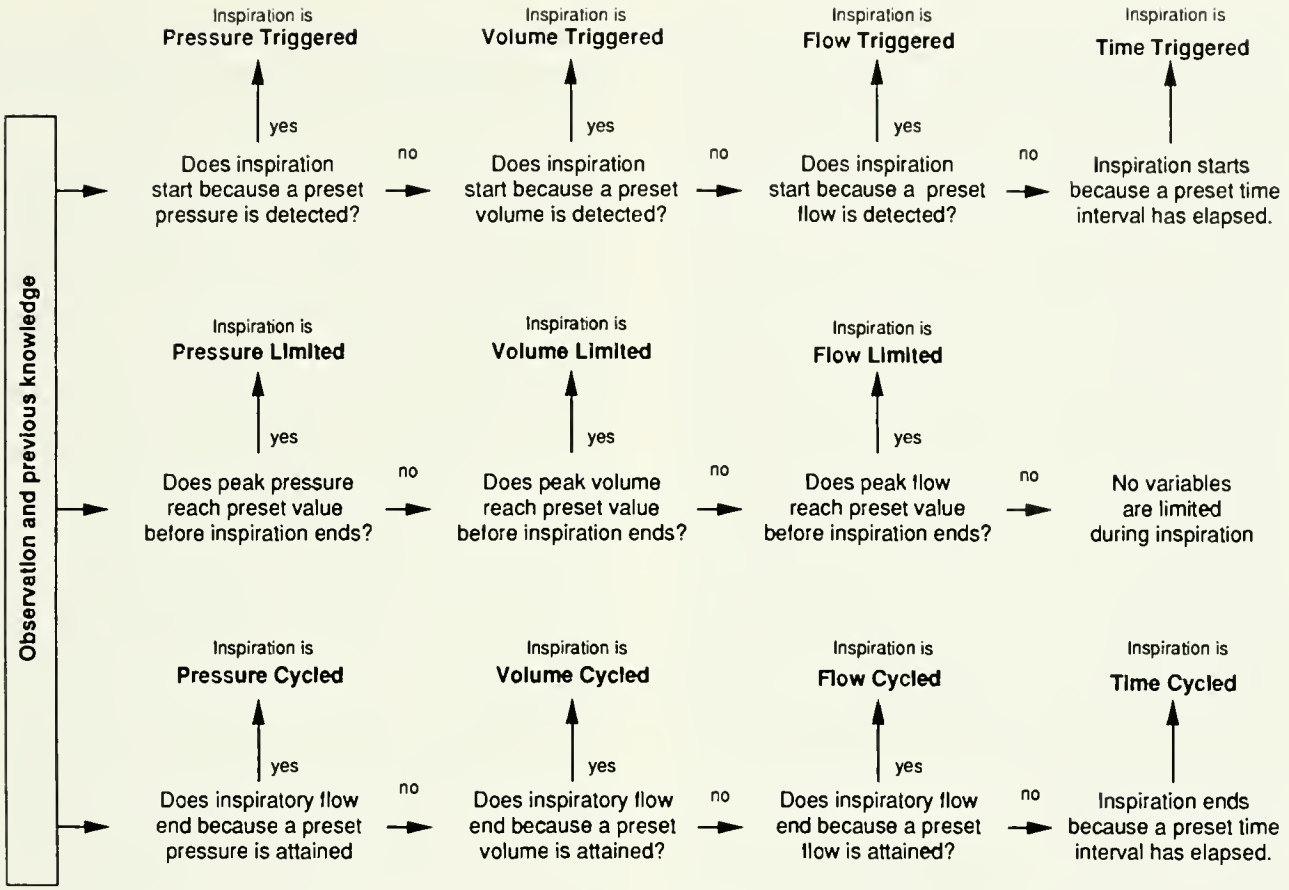

Fig. 7. Criteria for determining the phase variables during a ventilator-supported breath.

centage of the ventilatory period (defined by the set SIMV frequency) during which the ventilator switches from patient triggering to time triggering. Thus, a breath can be either mandatory or spontaneous, depending upon the coincidental occurrence of a patient effort within the timing window! A consideration of the pressure-support mode raises the question of whether setting an inspiratory pressure limit constitutes a mandatory breath. We need a more exact definition of the terms mandatory and spontaneous to help us to understand what the ventilator is doing, and to provide a basis for distinguishing among ventilatory modes.

To resolve these problems, we must recognize the importance of distinguishing among the types of breaths that can occur in any mode of ventilation. We must also use the simplest possible classification scheme in accordance with Ockham's Razor (or the Law of Parsimony), which states that the simplest explanation of a given phenomenon is the most correct.[25] This means that we should try to define as few types of breath as possible. One is clearly too few. Three or more are conceivable (eg, flow-controlled mandatory normal, flow-controlled mandatory sigh, and pressure-controlled spontaneous). Two seems about right—mandatory and spontaneous—based on the degree of control the patient can exercise.

Analysis should further illuminate the issue. When the patient is not connected to the ventilator, the brain determines the trigger, limit, and cycle values in response to physiologic signals supplied by chemoreceptors and the Hering-Breuer reflex. That is, the brain initiates a breath, it determines the peak inspiratory flowrate and waveform and the size of the tidal volume. Ventilatory failure can be thought of as a derangement of these values due to problems with the physiologic sensors, the brain's ability to process the signals, failure of the ventilatory muscles, or pathology of the lungs. An ideal ventilator would sense the body's physiologic gas exchange needs just as the brain does and would

provide assistance in proportion to the deficit in the trigger, limit, and cycle values.

Ventilatory failure can be partial or complete, giving rise to the need for partial or complete ventilatory support. In partial ventilatory failure, the body usually maintains a regular effort to trigger inspiration but has lost the capability to maintain adequate limit and cycle values (ie, peak inspiratory flowrate and tidal volume). The body tries to breathe spontaneously, and the ideal ventilator would sense and preserve this ventilatory drive by allowing patient triggering, limiting, and cycling of the assisted breath in exact proportion to the patient's need. (Unfortunately, sensor technology has not evolved enough to allow this level of sophistication, and we are forced to add the judgment of the ventilator operator into the control loop.) In full ventilatory failure, the body no longer attempts to trigger, limit, or cycle inspiration, and the ventilator must take over these functions. Thus, we can derive a clinically relevant definition of spontaneous and mandatory breaths.

Spontaneous breaths are those that the patient can both initiate and terminate with sufficient activity of the ventilatory muscles. If the ventilator determines either the start or end of inspiration, then the breath is considered to be mandatory. Figure 8 illustrates these definitions with an algorithm. Note that if the ventilator either time- or volume-cycles an inspiration, the breath is considered mandatory because it is terminated by the ventilator. However, if the ventilator flow-cycles an inspiration, it is not considered mandatory. The rate of decay of inspiratory flow is determined by the patient's lung mechanics and ventilatory muscle activity. Hence, pressure-limiting does not constrain inspiratory flowrate, and flow cycling does not necessarily dictate either the inspiratory time or the tidal volume. In other words, the ventilator attempts to match the patient's inspiratory demand, and it is really the patient who terminates the breath.

These definitions lead to the following examples: If the breath is triggered by the ventilator, then it is considered mandatory regardless of how it is limited and cycled. If the ventilator is set so that the patient may trigger a breath, but the inspiratory flowrate and tidal volume are preset (eg, assist/control or control mode), then the breath is mandatory because the patient cannot terminate the breath before or after the predetermined inspiratory time has elapsed. On the other hand, if the ventilator is set so that the patient can trigger a breath, but the inspiratory pressure is limited and the breath is flow cycled (eg, pressure-support mode), then the breath is spontaneous because the patient can still determine the instantaneous inspiratory flowrate and tidal volume depending on the activity of the ventilatory muscles.

The importance of defining breaths in this way is that it provides the basis for controlling the ventilator. Hence, it provides the basis for clinicians' understanding what the ventilator is doing.

Spontaneous breaths are typically supported either with some type of demand valve or with a continuous-flow circuit. With a demand valve, the ventilator performs work on the patient because a slight rise in airway pressure above baseline is required to shut it off (even if it is a clinically unimportant amount). With a continuous-flow system, fresh gas flows through the circuit at a constant rate, regardless of the patient's inspiratory flowrate. Thus, the patient must do work on the circuit to draw inspiratory gas. Therefore, when a demand valve is used to support spontaneous breaths, we say that spontaneous breaths are actively supported. When a continuous flow of fresh gas is

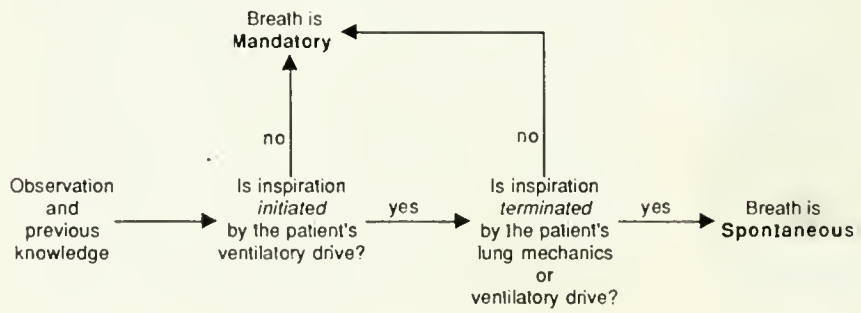

Fig. 8. Algorithm defining spontaneous and mandatory breaths. If the breath is *triggered* according to a preset frequency or minimum minute ventilation or *cycled* according to a preset frequency or tidal volume, the breath is *mandatory*. All other breaths are *spontaneous*.

used to entirely support spontaneous breaths, and no demand valve is used, we say that spontaneous breaths are passively supported.

2. Limit. Inspiratory time is defined as the time interval from the start of inspiratory flow to the start of expiratory flow. During inspiration, pressure, volume, and flow increase above their end-expiratory values. If one (or more) of these variables rises no higher than some preset value, we refer to the variable as a limit variable; but, we must distinguish the limit variable from the variable used to end inspiration (called a cycle variable). Therefore, we impose the additional criteria that inspiration is not terminated because a variable has met its preset limit value. In other words, a variable is limited if it increases to a preset value before inspiration ends. These criteria are illustrated in Figure 7. (Theoretically, a ventilator could also limit pressure, volume, and flow during expiration. In this case, the limit would be met before the next inspiration was triggered.)

For example, peak pressure during conventional or high-frequency oscillatory ventilation of neonates is frequently limited, but the preset pressure limit has nothing to do with the ending of inspiration. Rather, inspiration ends after a preset time interval (ie, the preset inspiratory time). Another example: When an inspiratory pause is used on a ventilator like the Bear 5, the volume delivered to the patient is limited to a preset value, but inspiration does not end with the delivery of that volume; rather, inspiration ends when a preset time interval (in this case, the pause time) has elapsed. One last example is the way peak inspiratory flowrate is often limited to a preset value (during ventilation of adults), but inspiration ends when a preset volume has been delivered.

Clinicians commonly and incorrectly use the terms limit and cycle interchangeably. This is encouraged by some ventilator manufacturers who use the term limit to describe what happens when a pressure-alarm threshold is met (ie, inspiration is terminated, and an alarm is activated). The term cycle, as defined later, is more appropriate in this situation.

In the equation of motion, pressure, volume, and flow are measured relative to their baseline values. Therefore, to maintain consistency, pressure, volume, and flow limits must be specified relative to their end-expiratory values. This is obvious for volume and flow with values of zero at end-expiration. However, there is currently confusion about pressure limits. For example, in the pressure-support mode, the pressure limit is specified as a value above end-expiratory pressure (ie, above PEEP). But the 'pressure limit' on an infant ventilator is measured relative to atmospheric pressure. Again, to maintain consistency, pressure limits should always be measured relative to the preset end-expiratory pressure. It is the pressure change above end-expiratory pressure that determines the tidal volume. Thinking of the pressure limit as being measured above atmospheric pressure (and using a ventilator that controls it as such) rather than above end-expiratory pressure leads to clinical misjudgments. For example, a common mistake made by clinicians inexperienced in infant ventilation is to increase the PEEP without increasing the peak inspiratory pressure limit and then to wonder why the P_{CO_2} increases.

Future microprocessor-controlled ventilators will undoubtedly measure and control volume. With a volume signal for feedback control (perhaps averaging two or three breaths), inspiratory flowrate or inspiratory time would be adjusted so that tidal volume delivery could be as accurate as flow measurements allow.

3. Cycle. Inspiration always ends (ie, is cycled off) because some variable has reached a preset value. The variable that is measured and used to terminate inspiration is called the cycle variable. The criteria for determining cycle variables are given in Figure 7.

Deciding which variable is used to terminate inspiration for a given ventilator can be confusing. For a variable to be used as a feedback signal (in this case, a cycling signal) it must first be measured. Most third-generation adult ventilators allow the operator to set a tidal volume and inspiratory flowrate, which would lead one to believe that the ventilator could be volume-cycled. However, closer inspection reveals that these ventilators do not measure volume. Rather, they determine the inspiratory time necessary to achieve the set tidal volume with the set inspiratory flowrate, making them

time-cycled. The tidal-volume dial can be thought of as an inspiratory time dial calibrated in units of volume rather than time.

Modern flow-controllers employ complex algorithms to deliver preset tidal volumes with a choice of flow waveforms. Being microprocessor-controlled, they actually control the instantaneous flowrate delivered by a proportional valve for discrete intervals of time. This means that once the tidal volume, peak inspiratory flowrate, and flow waveform have been selected, the microprocessor uses mathematical algorithms to set the duration, or length, of the flow waveform. The microprocessor then 'chops' the flow waveform into thin 'slices' of time, usually about 10 milliseconds in duration. During each slice of time, the ventilator actually delivers a constant flowrate, regardless of the selected waveform. This constant rate approximates the flow specified by the shape of the selected waveform at that particular moment in time. Thus, the microprocessor uses a series of stairsteps, both up and down, to digitally approximate the desired waveform.

We can visualize this process by a simple analogy. Imagine that inside the microprocessor is a series of file cabinets containing the required inspiratory flow-control information for every possi-ble tidal-volume setting (Fig. 9). Each cabinet contains drawers of cards representing the instan-taneous flowrate to be determined by the ventilator. The selected flow waveform determines in which column the required drawer is found, and the peak- flow setting determines the row. Opening the drawer, we find a series of cards. On each card is a value for instantaneous flow that the microprocessor uses to control the proportional valve for each 10-millisecond slice of inspiratory time. The values on the cards change according to the waveform. If a sinusoidal waveform has been selected, the flow values start at some baseline value, increase to the peak-flow setting, and fall again to the baseline value. The number of cards in the drawer depends on the inspiratory time and the microprocessor's clock rate. If, for example, the microprocessor can make changes in the proportional valve setting every 10 milliseconds and the inspiratory time is 2 seconds, there are 200 cards in the drawer.

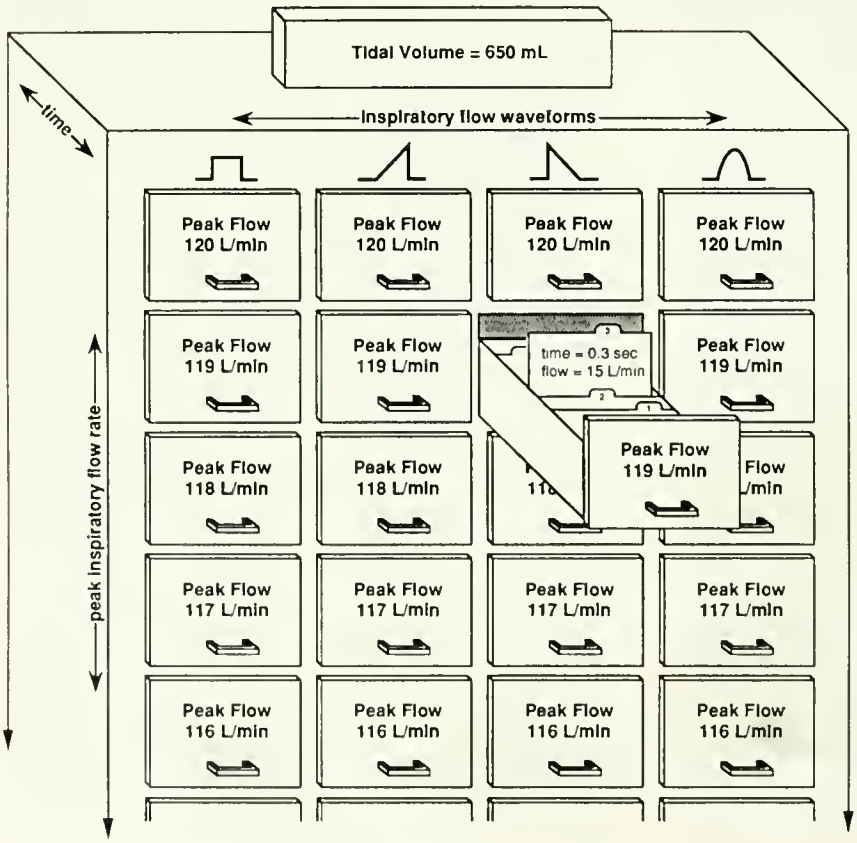

Fig. 9. File-cabinet analogy for flow control. For each tidal-volume setting, the operator selects the inspiratory flow waveform and peak inspiratory flowrate. The cards in each file drawer contain the value of inspiratory flow during each time interval (eg, each step lasts 10 milliseconds). The tidal volume is equal to the sum of the volumes represented by all the cards in a file drawer.

This analogy is accurate for the ideal, zero-load flow profile (with no patient connected). Once a load (the patient) is connected to the ventilator, the actual instantaneous flowrate is degraded because of the increasing airway pressure. When the ventilator detects that flow for one time slice falls below its preset card value, extra flow is added during the next slice to make up for the earlier lost flow. Compliance compensation schemes also change the card values from their original values. With each new breath, a new set of card values may be computed.

Some authors[5,14] have asserted that a ventilator can have 'mixed' cycling, which is contrary to the idea presented here that a ventilator can only control one variable at a time. The most common example given by these authors is a ventilator drive mechanism composed of a piston connected to a rod and a rotating crank. It is argued that one cannot distinguish time (ie, inspiratory time set by the frequency at which the crank rotates) or volume (ie, the stroke volume of the piston) as the cycling variable.

This problem can be addressed as follows: First of all, such a mechanism has been used in two general applications. One is as the drive mechanism of a high-frequency oscillator ventilator. In this application, for a given frequency and stroke volume of the piston, the pressure, volume, and flow delivered to the patient are usually the result of the relative impedances of the patient's respiratory system and the expiratory limb of the delivery circuit (usually configured as a variable low-pass filter). Here the pressure, volume, and flow waveforms delivered to the airway opening vary as respiratory-system mechanics change so that the ventilator is a time-controller as shown in Figure 6. Furthermore, it follows that inspiration does not end because a set pressure, volume, or flow has been achieved, leaving time as the only reasonable cycling variable. The same argument can be applied to a system in which a piston is used to compress a reservoir bag.

The second application is exemplified by ventilators like those of the Emerson series or the Bourns LS-104. With these ventilators, if the inspiratory time is set short enough and the tidal volume large enough, as the load imposed by a simulated patient increases, a point comes at which the motor is overloaded and cannot produce the desired settings. In this case, it becomes clear that inspiratory time is lengthened for as long as it takes the piston to reach the end of its stroke. Thus, time is 'sacrificed' for volume, and we must conclude that volume is the cycling variable.

4. Baseline. The significant characteristic of expiration is how the ventilator affects the way the control variables return to their baseline values. The variable controlled during the expiratory time is the baseline variable. Note that in the equation of motion, pressure, volume, and flow are measured relative to end-expiratory or baseline values and are thus initially all zero. Although the baseline value of any of these variables could theoretically be controlled, pressure control is the most practical and is implemented by all commonly used ventilators.

The ability of a ventilator to control the baseline variable means, for practical purposes, the ability to control expiratory transrespiratory pressure. Note that the emphasis is on transrespiratory pressure rather than airway pressure. This distinction is made first to accommodate the situation in which a negative-pressure ventilator is able to maintain a negative body-surface pressure during expiration. The baseline transrespiratory pressure difference (airway pressure – body surface pressure) in this case would be positive just as when conventional PEEP is used. However, end-expiratory airway pressure would be zero. Thus, thinking of PEEP in terms of airway pressure rather than transrespiratory pressure would be misleading.

The second reason is that it might be desirable to apply a negative transrespiratory pressure change to facilitate expiration in the face of increased airway resistance. Schulze et al[26] have described this technique as resistive unloading or negative ventilator resistance. (Actually, the term negative ventilator resistance is a misnomer because it is a technique for manipulating pressure, not resistance.) This is similar to the concept of using pressure support to decrease the resistive work of inspiration, except that the change in transrespiratory pressure is negative instead of positive, and an expiratory pressure limit is set below the baseline rather than

above it. More precisely, we set one baseline to assist expiratory flow and then another (when expiratory flow ceases) to restore FRC. This prevents gas trapping without the risk of alveolar collapse associated with negative end-expiratory pressure (NEEP). (Expiratory flow is driven by the pressure gradient [alveolar pressure - baseline airway pressure] created by inspiration [alveolar pressure = tidal volume/compliance]. Decreasing the baseline airway pressure during expiratory flow increases the pressure gradient and increases expiratory flow [flow = pressure gradient/resistance]. Conceptually, this effect is the same as decreasing expiratory resistance.)

We call the first baseline setting transient expiratory assist pressure (TEAP) and the second end-expiratory pressure (EEP). If the ventilatory frequency is set to zero, we can say that the patient is on a constant baseline pressure or constant airway pressure (CAP). TEAP, EEP, and CAP are all measured relative to body surface pressure. TEAP may be positive or negative, but EEP and CAP are usually positive.

There is another interpretation of baseline pressure: During high-frequency oscillatory ventilation, airway pressure takes on both positive and negative values relative to the mean airway pressure. In this context, baseline pressure is the mean airway pressure.

Perhaps we should digress for a moment to examine the concept of expiration. Expiratory time is defined as the interval from the start of expiratory flow to the start of inspiratory flow. Normally, expiratory flow terminates before the end of the expiratory time. If it does not (as can be determined by observing a flow waveform), then we know that gas trapping has occurred and alveolar pressure is higher than end-expiratory airway pressure.

Expiration is classified as being either active or passive. Active expiration occurs when either the expiratory muscles are active or the ventilator generates a negative transrespiratory pressure that drives flow in the expiratory direction. Use of the resistive unloading technique described earlier is an example of active expiration caused by the ventilator. Another example of ventilator-caused active expiration is in the use of high-frequency oscillatory ventilation. Because a high-frequency oscillator generates a cycle of positive and negative pressure at the airway opening, gas flow in the expiratory direction is augmented. Passive expiration occurs when expiratory flow is caused only by the pressure due to the elastic recoil of the respiratory system.

D. Conditional Variables

Figure 5 illustrates that for each breath there is a specific pattern of control and phase variables. The ventilator may either keep this pattern constant for each breath, or it may introduce other patterns (eg, one for mandatory and one for spontaneous breaths). Some ventilators are capable of complex patterns such as two types of mandatory breaths (one normal, one sigh) and two types of spontaneous breaths (eg, with two different pressure limits). In essence, the ventilator must decide which pattern of control and phase variables to implement before each breath, depending on the value of some preset conditional variable(s). A simple example is the Bennett MA-1 in the control mode. Each breath is time triggered, flow limited, and volume cycled. The trigger, flow, and volume variables have preset values (eg, frequency 20 cycles/min, inspiratory flow 60 L/min, and tidal volume 750 mL). However, every few minutes a sigh breath is introduced that has a different set of phase variable values (eg, frequency 2 sighs every 15 minutes, tidal volume 1,500 mL). How does the ventilator know to do this? Conceptually, we can say that before each breath pattern is selected, the ventilator examines the value of some conditional variable to see if it has reached a preset threshold value. If the threshold has been met, one pattern is selected: if not, another pattern is selected. In the case of the MA-1, the conditional variable is time—once a preset time interval has elapsed the ventilator switches to the sigh pattern.

Another example is the SIMV mode. If the ventilator detects a patient effort and the SIMV 'window' is open, a spontaneous breath is delivered. If an effort is not sensed and the window has closed, a mandatory breath is delivered. Two conditional variables, pressure (or flow) and time are present.

Yet another example is the mandatory minute ventilation mode in which the conditional variable is exhaled minute ventilation. When minute ven-

tilation falls below a preset threshold, the ventilator may switch from a spontaneous breath pattern to a mandatory pattern (eg, the Bear 5), or it may change the parameters of the spontaneous breath (eg, the Hamilton Veolar increases the pressure limit). In the future, ventilators may be able to switch patterns based on arterial blood gas values. There may be several different conditional variables, different combinations of which will result in a variety of breath patterns within each mode.

To many clinicians, a mode is just a label on a ventilator's control panel, and clinicians appear to associate modes with various clinical conditions (eg, "pressure support is good for weaning"). Modes are often viewed as distinct ventilatory methods that have little in common with each other. Manufacturers enforce this view by creating new names and acronyms (or adopting those of prominent researchers) and more labels for the control panel. Unfortunately, this approach has become awkward and confusing when applied to modern ventilators. It encourages a narrow view of ventilator performance that limits our ability to manage the vast array of clinical problems. I suggest an approach that emphasizes similarities rather than differences.

In the first place, a mode of ventilation is no more than a shorthand notation, or jargon, that represents a specific set of characteristics of particular importance to the clinician. In defining modes, authors sometimes resort to rambling descriptions of how the ventilator and patient interact, but in no particular order and with no unifying theme. This makes recall difficult. I believe that a better approach is to realize that a mode of ventilation is best defined as a particular set of control and phase variables. In particular, the control and phase variables for mandatory and spontaneous breaths must be specified. Using this approach, modes can be conveniently defined as shown in Table 2.

Examination of Table 2 reveals an interesting fact: The commonly used mode names are not very specific in describing a given pattern of ventilation. For example, assist/control can mean that inspiration is pressure triggered, flow limited, and volume cycled; or volume triggered, flow limited, and time cycled; or pressure triggered, volume limited, and time cycled. But, it has not always been this way. When the Bennett MA–1 was the most common ventilator, assist/control was understood to mean simply that the tidal volume was preset and inspiration was pressure triggered on every breath (ie, the breath was either pressure or time triggered, flow limited, and volume cycled). Our traditional concepts do not fit today's more flexible technology. And, the situation promises to get worse with future ventilators.

So what do we do about the proliferation of new modes? Clearly more labels will only make things worse. I suggest that clinicians focus not on modes but rather on specific patterns of control and phase variables. Of course, this requires that ventilator manufacturers provide machines that allow this degree of control and have appropriate control-panel labeling. I envision a machine that allows the clinician to select the conditional, control, and phase variables for both mandatory and spontaneous breaths. Such innovation probably will not come at the request of clinicians because many clinicians lack awareness of the scope of the problem, have not articulated a solution, and are not well organized as a consumer group. However, if a manufacturer had the courage to lead in this direction, clinician acceptance would make or break the product. Acceptance follows on the heels of understanding. I hope that this paper stimulates the creativity required on both sides of the issue.

IV. Output

Just as the study of cardiovascular physiology involves the study of electrocardiograms and blood pressure waveforms, the study of ventilator operation requires the examination of output waveforms. The waveforms of interest, of course, are the pressure, volume, and flow waveforms we have used throughout this discussion.

Output waveforms are graphed in groups of three (Fig. 10) in the conventional order of presentation—pressure, volume, and flow, based on the mathematical convention used for the equation of motion, a specific example of a first-order linear differential equation. Convention also dictates that positive values (above the horizontal axis) correspond to inspiration, and negative values (below the horizontal axis) correspond to expiration. The horizontal axes of all three graphs are the same and

Table 2. Variables for Mandatory and Spontaneous Breaths in Commonly Used Ventilatory Modes. A Given Mode May Apply to Several Patterns of Phase Variables, Depending on Ventilator and How It Is Used

	Mandatory				Spontaneous			
Mode	Control	Trigger*	Limit	Cycle	Control	Trigger	Limit	Cycle
Control	flow†	time	volume flow	volume time	NA‡	NA	NA	NA
A/C or CMV	flow	pressure volume time	volume flow	volume time	NA	NA	NA	NA
IMV (continuous flow)	pressure flow	time	volume flow	volume time	—	—	—	—
SIMV (continuous flow)	pressure flow	pressure volume flow time	pressure volume flow	volume time	—	—	—	—
SIMV (demand flow)	pressure flow	pressure volume flow time	pressure volume flow time	time	pressure flow	pressure	pressure	pressure
PS	—	—	—	—	pressure	pressure	pressure	flow
PS + SIMV	pressure flow	pressure volume flow time	pressure volume flow time	time	pressure	pressure flow	pressure	flow
CAP or CPAP (continuous flow)	—	—	—	—	pressure	—	pressure	—
CAP or CPAP (demand flow)	—	—	—	—	pressure	pressure flow	pressure	—
PC	pressure	time	pressure	time	NA	NA	NA	—

* Whether a breath is patient triggered depends on the sensitivity setting and the magnitude of the patient's inspiratory effort.
† For the purposes of this table, flow control is equivalent to volume control. Baseline PEEP is assumed to be available for all modes.
‡ NA = not applicable; A/C = assist/control; CMV = continuous mandatory ventilation; IMV = intermittent mandatory ventilation; — = the ventilator does not respond; PS = pressure support; SIMV = synchronized mandatory ventilation; CAP = constant airway pressure; CPAP = continous positive airway pressure; PC = pressure control.

have the units of time. The vertical axes are in units of the measured variables. The ventilator determines the shape of the control waveform, and the shapes of the other waveforms are determined by the compliance and resistance of the load according to the equation of motion. For the purpose of identifying output waveforms, the specific baseline values of each variable are irrelevant. Therefore, the origin of the vertical axis is labeled zero. What is important is the relative magnitude of each of the variables and how the value of one affects or is affected by the value of the others.

Remember that the waveforms used to define categories of ventilator outputs are idealized. That is, they are precisely defined by mathematical equations and are meant to characterize the opera-

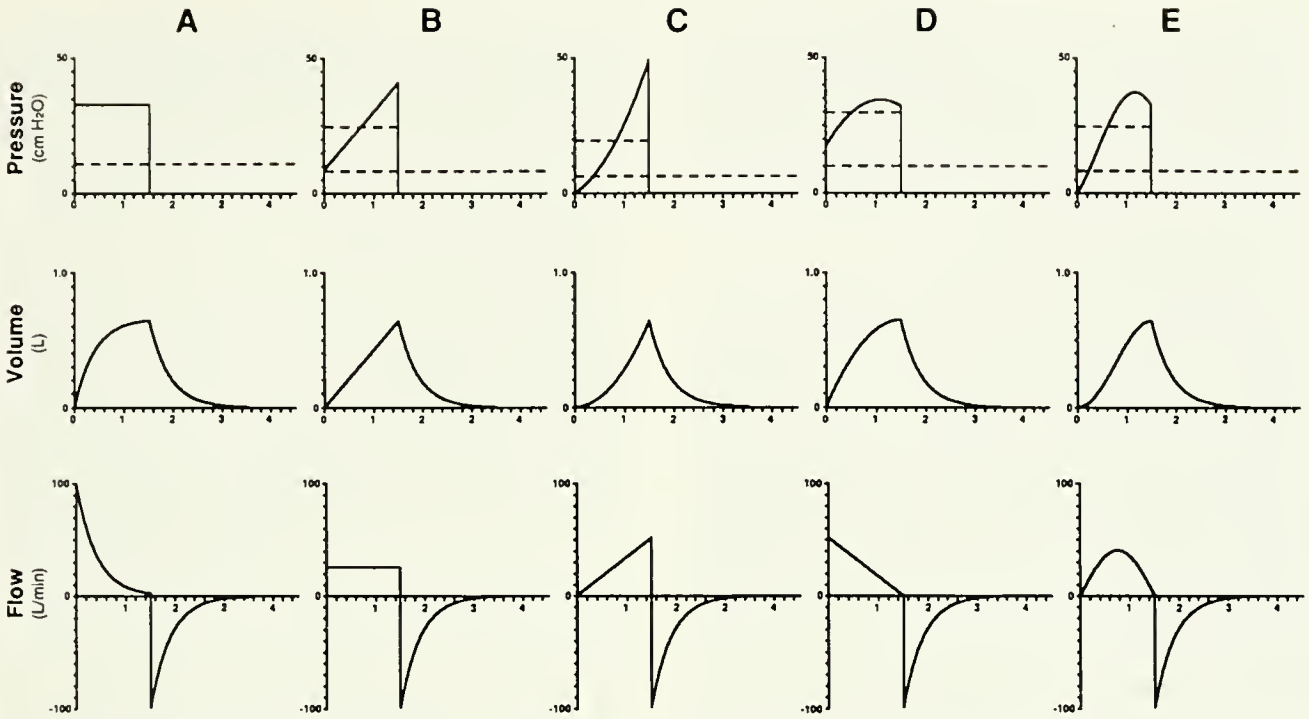

Fig. 10. Theoretical output waveforms for A—pressure-controlled inspiration with rectangular pressure waveform, identical to flow-controlled inspiration with an exponential-decay flow waveform; B—flow-controlled inspiration with rectangular flow waveform, identical to volume-controlled inspiration with an ascending-ramp volume waveform; C—flow-controlled inspiration with an ascending-ramp flow waveform; D—flow-controlled inspiration with a descending-ramp flow waveform; and E—flow-controlled inspiration with a sinusoidal flow waveform. The short dashed lines represent mean inspiratory pressure while the longer dashed lines denote mean airway pressure (assuming zero end-expiratory pressure). For the rectangular pressure waveform in A, the mean inspiratory pressure is the same as the peak inspiratory pressure. These output waveforms were created by (1) defining the control waveform (eg, an ascending-ramp flow waveform is specified as flow = constant × time) and specifying that tidal volume equals 644 mL (about 9 mL/kg for a normal adult); (2) specifying the desired values for resistance and compliance (for these waveforms, compliance = 20 mL/cm H_2O and resistance = 20 cm $H_2O \cdot s \cdot L^{-1}$, according to ANSI recommendations[9]); (3) substituting the above information into the equation of motion; and (4) using a computer to solve the equation for pressure, volume, and flow and plotting the results against time.

tion of the ventilator's control system. As such, they do not show the minor deviations or 'noise' often seen in waveforms recorded during actual ventilator use. These waveform imperfections can be caused by a variety of extraneous variables such as vibration and turbulence, and the appearance of the waveform is affected by the scaling of the time axis. The waveforms also do not show the effects of the resistance of the expiratory side of the patient circuit, which vary depending on the ventilator and type of circuit.

No ventilator is an ideal controller, and ventilators are designed to only approximate a particular waveform. Idealized or standard waveforms are nevertheless helpful because they are common in other fields (eg, electrical engineering), which makes it possible to use mathematical procedures and terminology that have already been developed. For example, a standard mathematical equation is used to describe the most common waveforms for each control variable. This known equation may be substituted into the equation of motion, which is then solved to obtain the equations for the other two variables. Once the equations for pressure, volume, and flow are known, they are easily graphed. This is the process used to generate the graphs in Figure 10.

As mentioned previously, most ventilator waveforms can be classified as rectangular, exponential, ramp, or sinusoidal (including sigmoidal and oscil-

lating). Although a variety of subtypes are possible, only the most common are presented here and are listed according to the shape of the control-variable waveform. Any new waveforms produced by future ventilators can easily be accommodated by this system.

A. Pressure Waveforms

1. Rectangular. A rectangular waveform is referred to mathematically as a step or instantaneous change in transrespiratory pressure from one constant value to another. In response, volume rises exponentially from zero to a steady-state value equal to compliance × the change in airway pressure, or peak inspiratory pressure – PEEP (ie, PIP – PEEP). Inspiratory flow falls exponentially from a peak value (at the start of inspiration) equal to (PIP - PEEP)/resistance.

2. Exponential. Exponential pressure waveforms are commonly used during neonatal ventilation. Ventilators such as the Bear Cub are designed to deliver a modified rectangular waveform that typically results in a gradual rather than instantaneous change in pressure at the start of inspiration. Thus, depending upon the specific ventilator settings (eg, short inspiratory time, low flowrate, and high PIP), the pressure waveform may never attain a constant value and resembles an exponential curve instead. In response, the volume and flow waveforms are also exponential, but their peak values are less than with a rectangular pressure waveform.

3. Sinusoidal. A sinusoidal pressure waveform can be created by attaching a piston either to a rotating crank or to a linear-drive motor driven by an oscillating signal generator. In response, the volume and flow waveforms are also sinusoidal, but they attain their peak values at different times.

4. Oscillating. Oscillating pressure waveforms can take on a variety of shapes from sinusoidal (Mira Hummingbird) to ramp (SensorMedics 3100 oscillator) to roughly triangular (Infrasonics Star Oscillator). The distinguishing feature of a ventilator classified as an oscillator is that it can create negative transrespiratory pressure (that is, if the mean airway pressure is set equal to atmospheric pressure, then the airway pressure waveform oscillates above and below zero). If the pressure waveform is sinusoidal, volume and flow also are sinusoidal but out of phase with each other (ie, their peak values occur at different times). Other waveforms produce more complex volume and flow waveforms.

B. Volume Waveforms

1. Ramp. Volume controllers that produce an ascending-ramp waveform (eg, the Bennett MA–1) produce a linear rise in volume from zero at the start of inspiration to the peak value (ie, the set tidal volume) at end-inspiration. In response, the flow waveform is rectangular in shape. The pressure waveform rises instantaneously from zero to a value equal to resistance times flow at the start of inspiration, from which it rises linearly to its peak value (ie, PIP) equal to

$$PIP = \frac{tidal\ volume}{compliance} + (flow)(resistance) + PEEP. \quad [2]$$

2. Sinusoidal. This waveform is most often produced by ventilators driven by a piston attached to a rotating crank (eg, Engstrom and Emerson ventilators). The output waveform of this type of ventilator is approximated by the first half of a cosine curve (in this case, sometimes referred to as a sigmoidal curve). Because the volume is sinusoidal during inspiration, pressure and flow are also sinusoidal.

C. Flow Waveforms

1. Rectangular. A rectangular flow waveform is perhaps the most common output. When the flow waveform is rectangular, volume has a ramp waveform, and pressure is a step followed by a ramp as described for the ramp volume waveform.

2. Ramp. The ramp waveform is what many respiratory care practitioners (and ventilator manufacturers) call an accelerating or decelerating waveform. The term ramp is borrowed from electronic engineering and is preferred for three reasons. First, the name ramp gives a more obvious visual image of the actual shape of the waveform.

Second, the term ramp has been described mathematically and used universally for much longer than mechanical ventilators have been in existence. Third, the analogy of something accelerating or decelerating is misapplied. For example, when a car is moving, we say it has a certain speed (ie, speed = Δdistance/Δtime). If the speed increases with time, we say that the car accelerates (ie, acceleration = Δspeed/Δtime), not that the speed accelerates. The speed of moving gas is expressed as a flowrate (ie, flowrate = area of tube × Δdistance/Δtime). If the flowrate increases, we properly say that the gas accelerates (ie, acceleration = Δflowrate/Δtime), not that the flow accelerates. In scientific terms "The acceleration of a particle is the rate of change of its velocity with time."[27]

a. Ascending. A true ascending ramp waveform starts at zero and increases linearly to the peak value. Ventilator flow waveforms are usually truncated: inspiration starts with an initial instantaneous flow (eg, the Bear 5 starts inspiration at 50% of the set peak flow). Flow then increases linearly to the set peak flowrate. In response to an ascending-ramp flow waveform, the pressure and volume waveforms are exponential with an upward concave shape.

b. Descending. A true descending-ramp waveform starts at the peak value and decreases linearly to zero. Ventilator flow waveforms are usually truncated; inspiratory flowrate decreases linearly from the set peak flow until it reaches some arbitrary threshold at which flow drops immediately to zero (eg, the Bennett 7200a ends inspiration when the flowrate drops to 5 L/min). In response to a descending-ramp flow waveform, the pressure and volume waveforms are exponential with a concave downward shape.

3. Sinusoidal. Some ventilators offer a mode in which the inspiratory flow waveform approximates the shape of the first half of a sine wave. As with the ramp waveform, ventilators often truncate the sine waveform by starting and ending flow at some percentage of the set peak flow rather than starting and ending at zero flow. In response to a sinusoidal flow waveform, the pressure and volume waveforms are sinusoidal but out of phase with each other.

D. Effects of the Patient Circuit

So far in this discussion, I have implied that what comes out of the ventilator is the same as what goes into the patient. However, pressure, volume, and flow measured inside the ventilator are never the same as pressure, volume, and flow measured at the patient's airway opening. The reason, of course, is that the patient circuit has its own compliance (actually, the compliance of the tubing material plus the compressibility of the inspired gas) and resistance. Therefore, the pressure measured inside the ventilator on the inspiratory side (eg, on a Bennett MA-1) is always higher than the pressure at the airway opening because of the elastic and flow-resistive pressure drops created by the patient circuit. Volume and flow coming out of the ventilator are always higher than the volume and flow delivered to the patient because of the effect of the compliance of the patient circuit. This compliance effect absorbs both volume and flow.

It can be shown by analogy to electrical circuits that the compliance of the delivery circuit is connected in parallel with the compliance of the respiratory system (that is, pneumatic compliance is analogous to electrical capacitance, and pneumatic resistance is analogous to electrical resistance).[9] Therefore, the total compliance of the ventilator-patient system is simply the sum of the two compliances. In a similar manner, the resistance of the delivery circuit is shown to be connected in series with the respiratory-system resistance so that the total resistance is the sum of the two. From these assumptions, it can be shown that the relation between the volume input to the patient (at the point of connection to the patient's airway opening) and the volume output from the ventilator (at the point of connection to the patient circuit) is described by

$$\textit{volume input to patient} = \left(\frac{1}{1 + \frac{C_{pc}}{C_{rs}}}\right)(\textit{volume output from ventilator}), \quad [3]$$

where C_{pc} is the compliance of the patient circuit and C_{rs} is the total compliance of the patient's respiratory system.

The equation shows that the greater the patient-circuit compliance is compared to the patient's res-

piratory system, the greater the denominator on the right-hand side of the equation and hence the smaller the delivered tidal volume compared to the volume coming out of the ventilator's drive mechanism.

Assuming that the volume exiting the ventilator is the set tidal volume, the patient-circuit compliance is calculated as

$$C_{pc} = \frac{V_{T-set}}{P_{plateau} - EEP}, \quad [4]$$

where $P_{plateau}$ is the pressure measured during an inspiratory-hold maneuver with the Y-adapter of the patient circuit occluded (patient not connected).

Some authors recommend the use of PIP for $P_{plateau}$ in the above equation. This can lead to a slight underestimation of patient circuit compliance. $P_{plateau}$ is slightly lower than PIP because of the flow-resistive pressure drop of the patient circuit (if PIP is measured inside the ventilator on the inspiratory side). This difference is greatest in small-bore, corrugated, patient-circuit tubing but is probably not clinically important.

The effects of patient-circuit compliance are most troublesome during volume-controlled ventilation. For example, with neonates, the patient-circuit compliance can be as much as three times that of the respiratory system, even with small-bore tubing and a small-volume humidifier. Thus, the volume delivered to the patient may be as little as 25% of that coming from the ventilator, with 75% of the volume compressed in the patient circuit.

You can see that the patient circuit has the same magnitude of effect on mean inspiratory flowrate by dividing "volume input to patient" and "volume output from ventilator" in Equation 3 by inspiratory time. The discrepancy between the set and delivered tidal volume and flow must be taken into account when using most ventilators. However, some, like the Bennett-7200 series, make the appropriate calculations and adjustments automatically.

During pressure-controlled ventilation, the compliance of the patient circuit has the effect of rounding the leading edge of a rectangular pressure waveform, which could possibly reduce the volume delivered to the patient. This effect is avoided if the pressure limit is maintained for at least five respiratory-system time constants. (The time constant is a measure of the time required for the passive respiratory system to respond to abrupt changes in ventilating pressure.[9,18])

For both pressure- and volume-controlled ventilation, the patient-circuit compliance and resistance increase the expiratory time constant. Thus, a high circuit compliance coupled with a short expiratory time can lead to inadvertent positive end-expiratory pressure (ie, auto-PEEP).

Remember that, in general, the set values for pressure, volume, and flow differ from the output (from ventilator) values because of calibration errors and from the input (to the patient) values because of the effects of the patient circuit. Thus, there are two general sources of error that cause discrepancies between the desired and actual patient values.

V. Alarm Systems

Ventilator alarms have also increased in number and complexity. Fortunately, the system we have been using to classify ventilators is easily adaptable to alarms.

Day and MacIntyre[28] have stressed that the goal of ventilator alarms is to warn of *events*. They define an event as any condition or occurrence that requires clinician awareness or action. Technical events are those involving an inadvertent change in the ventilator's performance; patient events are those involving a change in the patient's clinical status.[28] A ventilatof may be equipped with any conceivable vital sign monitor, so the appropriate scope of surveillance needs to be defined. The most logical scope would include the ventilator's mechanical-electronic operation and those variables associated with the mechanics of breathing (ie, pressure, volume, flow, and time). Because the ventilator is in intimate contact with exhaled gas, it seems appropriate to include the analysis of ex-

Table 3. A Four-Level Classification of Ventilator Alarms: Level 1 Indicates Critical Ventilator Malfunction, Level 2 Noncritical Ventilator Malfunction (Not Immediately Life Threatening), Level 3 Patient Status Change, Level 4 Operator Alert (Inappropriate Control Setting or Alarm Threshold)

	Level 1	Level 2	Level 3	Level 4
Alarm Characteristics				
Mandatory?	yes	yes	no	yes
Redundant?	yes	no	no	no
Non-cancelling?	yes	no	no	yes
Audible?	yes	yes	yes	no
Visual?	yes	yes	yes	yes
Automatic backup response?	yes	no	no	no
Automatic reset:				
Audible?	yes	yes	yes	yes
Visual?	no	yes	yes	yes
Applicable Alarm Categories				
Input:				
Electric power?	yes	no	no	no
Pneumatic power?	yes	no	no	no
Control circuit:				
Inverse I:E?	no	yes	no	yes
Incompatible settings?	no	no	no	yes
Mechanical/electronic fault?	yes	no	no	no
Output:				
Pressure?*	yes	yes	yes	yes
Volume?†	yes	yes	yes	yes
Flow?‡	yes	yes	yes	yes
Minute ventilation?	yes	yes	yes	yes
Time?§	yes	yes	yes	yes
Inspired gas (F_{IO_2}, temperature)?	yes	yes	no	yes
Expired gas (F_{EO_2}, F_{ECO_2})?¶	no	no	yes	no

*Pressure alarms = high & low peak, mean, and baseline.
†High & low inhaled & exhaled tidal volume. May include alarm for leak.
‡Alarm triggered if expiratory flow does not fall below set threshold (gas trapping).
§ Inspiratory or expiratory times too long or too short.
¶ May include tracer gases for functional residual capacity calculation.

haled gas for oxygen and carbon dioxide concentrations. Other devices, such as hemodynamic and cardiac monitors and pulse oximeters seem less appropriate for inclusion in ventilator design.

Alarms may be audible, visual, or both, depending upon the seriousness of the alarm condition. Visual alarms may be as simple as colored lights or as complex as alphanumeric messages to the operator indicating the exact nature of the fault condition. Specifications for an alarm event should include (1) conditions that trigger the alarm, (2) the alarm response in the form of audible and/or visual messages, (3) any associated ventilator response such as termination of inspiration or failure to operate, and (4) whether the alarm must be manually reset or resets itself when the alarm condition is rectified. Table 3 outlines the various levels of alarm priority along with characteristics and appropriate categories. Alarm categories are based on the ventilator classification scheme.

A. Input Power Alarms

1. Loss of electrical power. Most ventilators have some sort of battery backup in case of electrical power failure, even if the batteries only power alarms. Ventilators typically have alarms that are activated if the electrical power is cut off while the machine is still switched on (eg, if the power cord is accidentally pulled out of the wall socket). If the ventilator is designed to operate on battery power (eg, transport ventilators), an alarm usually warns of a low-battery condition.

2. Loss of pneumatic power. Ventilators that use pneumatic power have alarms that are activated if either the oxygen or air supply is cut off or reduced below some specified driving pressure. In some cases, the alarm is activated by an electronic pressure switch (eg, Bennett 7200); however, in others, the alarm is pneumatically operated as a part of the blender (eg, Siemens Servo 900C).

B. Control Circuit Alarms

Control circuit alarms are those that either warn the operator that the set control variables are incompatible (eg, inverse I:E) or that some aspect of a ventilator self-test has failed. In the latter case, there may something wrong with the ventilator control circuitry itself (eg, a microprocessor failure), and the ventilator generally responds with some generic message like "ventilator inoperative."

C. Output Alarms

Output alarms are those triggered by an unacceptable state of the ventilator's output. More specifically, an output alarm is activated when the value of a control variable (pressure, volume, flow, or time) falls outside an expected range. Some possibilities include:

1. Pressure.

a. High and low peak airway pressure—indicating a possible endotracheal tube obstruction or leak in the patient circuit, respectively.

b. High and low mean airway pressure—indicating a possible leak in the patient circuit or a change in ventilatory pattern that might lead to a change in the patient's oxygenation status (ie, within reasonable limits, oxygenation is roughly proportional to mean airway pressure).

c. High and low baseline pressure (PEEP or CPAP)—indicating a possible patient-circuit or exhalation-manifold obstruction (or inadvertent PEEP), and disconnection of the patient from the patient circuit, respectively.

d. Failure of airway pressure to return to baseline within a specified period—indicating a possible patient-circuit obstruction or exhalation-manifold malfunction.

2. Volume. High and low exhaled tidal volume, indicating changes in respiratory-system time constant during pressure-controlled ventilation or possible disconnection of the patient from the patient circuit.

3. Flow. High and low exhaled minute ventilation indicating hyperventilation (or possible machine self-triggering) and possible apnea or disconnection of the patient from the patient circuit, respectively.

4. Time.

a. High or low ventilatory frequency—indicating hyperventilation (or possible machine self-triggering) and possible apnea, respectively.

b. Inspiratory time too long or too short—too long, indicating a possible patient-circuit obstruction or exhalation-manifold malfunction; too short, indicating that adequate tidal volume may not have been delivered (in a pressure-control mode) or that gas distribution in the lungs may not be optimal.

c. Expiratory time too long or too short—too long, indicating apnea; too short, warning of alveolar gas trapping (ie, expiratory time should be ≥ five time constants of the respiratory system).

5. Inspired gas.

a. High/low inspired gas temperature.
b. High/low F_{IO_2}.

6. Expired gas. Because ventilators are designed to control the mechanical results of exhalation, they

may be easily adapted to the analysis of exhaled gas, and alarms may be set for specific limits.

a. Exhaled carbon dioxide tension. End-tidal carbon dioxide monitoring may reflect P_{aCO_2} and thus indicate the level of ventilation. Mean exhaled P_{CO_2} coupled with minute ventilation measurements could provide information about CO_2 production ($\dot{V}_{CO_2}$) and contribute to the calculation of the respiratory exchange ratio and the tidal volume-dead space ratio (V_D/V_T).

b. Exhaled oxygen tension.[29] Analysis of end- tidal and mean exhaled P_{O_2} may provide information about gas exchange and could be used with CO_2 data to calculate the respiratory exchange ratio.

How To Speak Machine Language

A successful classification scheme should easily accommodate the differing needs and interests of its intended audience. An administrator may need to know only where and for what type of patient the ventilator can be used (making the input power and control variables important). The clinician needs to know how the ventilator is operated (making phase variables and output characteristics of interest). The student, educator, or researcher may need to understand the internal workings of the ventilator (requiring the knowledge of drive mechanism, compressor motor-linkage, output control valves, and control variables and waveforms). Certainly, the manufacturer's representatives should be conversant with all of this information and be able to provide it to interested parties. The classification system presented here is designed to meet these needs in a logical, consistent, and unambiguous manner. Let us explore some ways that it could be used to communicate ideas in ventilator lingo.

The average clinician needs to describe very little about a ventilator during the course of the daily routine. Hence, the terms volume ventilator (allowing a preset volume) and pressure ventilator (allowing a preset inspiratory pressure) are often heard. However, confusion quickly arises when one attempts to describe how a ventilator operates, especially when one is speaking to someone who has little experience. Terms like pressure generator and flow generator are often used but may be ambiguous because all ventilators generate pressure, volume, and flow all at once. The idea one would like to convey is that a ventilator controls a particular variable, hence the terms pressure-controller, volume-controller, and flow-controller are more specific. The outline approach to classification lends itself to varying degrees of detail. For example, an Emerson Iron Lung can be described as an electrically powered, pressure controller that produces a sinusoidal pressure waveform. A more detailed description adds that it is (1) time triggered, (2) time cycled, (3) provides no baseline pressure control, and (4) uses an electrically controlled drive mechanism consisting of a diaphragm, rotating crank, and connecting rod driven by an electric motor. It operates in the control mode only and has no alarms. A more complex ventilator like the Bennett 7200 can be described as an electrically powered, pressure- or flow-controller. A more detailed description includes the points that it (1) is triggered by time, pressure, flow, or hand; (2) is pressure or volume limited; (3) is time or flow cycled; (4) controls baseline pressure (PEEP/CPAP); and (5) uses an electronic (microprocessor) control circuit and a drive mechanism/output-control valve consisting of a pressure regulator and proportional valve with a pneumatic-diaphragm exhalation-manifold valve. One could go on and list the multitude of operating modes and alarms.

Needless to say, the more complicated the ventilator, the more involved the description. Perhaps more commonly, clinicians need to refer to a mode of ventilation rather than to a specific ventilator. The most general terms seem to be volume- versus pressure-controlled ventilation. The term pressure-controlled ventilation should cause no problems, but use of the term volume-controlled ventilation may be confusing. A strict interpretation of the classification system presented in this discussion requires a distinction between volume-controlled and flow-controlled modes. However, in the interest of convenience, the term volume-controlled is acceptable for both. Any breath that is directly flow controlled is indirectly volume controlled and vice versa (but when describing a ventilator rather than a mode of ventilation, you should make the distinction between volume and flow control). More information about the mode of ventilation can be

conveyed by stating the control and phase variables for mandatory and spontaneous breaths, as illustrated in Table 2. In fact, you can communicate most of the important information by specifying the phase variables for the mandatory and spontaneous breaths. For example, a description of SIMV with pressure support might be mandatory breaths are pressure triggered, flow limited, and time cycled; spontaneous breaths are pressure triggered, pressure limited, and flow cycled. What more do you need?

Use of this system may be uncomfortable at first, especially to those who are already accustomed to some other system. But, if the basic ideas are learned, the rest is easy. It is similar to the way that the game of checkers—with only a few simple rules—leads to an almost infinite variety of playing strategies and tactics. But if two people playing the game do not know and use the same set of rules, confusion and conflict result. To carry the analogy further, the person who sees the similarity among patterns of checker position is much more likely to be successful at the game than the player who sees each position as different and unique. In the same way, the clinician who sees the similarity of design features and applies the same operational definitions to all ventilators more easily understands ventilators and modes of ventilation than the clinician who tries to memorize endless lists of different features. In the words of Blaise Pascal,

> *If you make people think they are thinking, they will love you. But if you really make them think, they will hate you.*

ACKNOWLEDGMENTS

I could not have developed the ideas presented in this manuscript without the meticulous and patient guidance of Frank P Primiano Jr PhD and Warren G Sanborn PhD. Their insightful contributions are much appreciated.

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GLOSSARY

Active expiration—expiration assisted by the ventilatory muscles or by a ventilator-induced change in transrespiratory pressure causing flow in the expiratory direction.

Alarm event—any condition or occurrence that requires clinician awareness or action.

Assisted ventilation—the act of continuously assisting inspiration and/or expiration.

Assisted expiration—expiratory flow generated by a negative change in transrespiratory pressure due to an external agent (ie, a drop in airway pressure below baseline).

Assisted inspiration—inspiratory flow generated by a positive change in transrespiratory pressure due to an external agent (ie, a rise in airway pressure above baseline).

Closed-loop control—a control scheme in which the actual output of a system is measured and compared to the desired output. If there is a difference caused by external disturbances, the actual output is modified to bring it closer to the desired output.

Compressor—a device whose internal volume can be changed to increase the pressure of the gas it contains.

Constant airway pressure (CAP)—a therapeutic modality that maintains a constant (usually positive) transrespiratory pressure. CAP is not a ventilatory mode because it does not generate a tidal volume, and hypoventilation will occur if the patient becomes apneic.

Control circuit—the ventilator subsystem responsible for controlling the drive mechanism and/or the output control valves.

Control variable—the variable (either pressure, volume, flow, or time) that the ventilator manipulates to cause inspiration. This variable is identified by the fact that its behavior remains consistent despite changes in ventilatory load.

Cycle—*verb*, to end a mechanically supported inspiration.

Demand valve—an output control valve that turns inspiratory flow on and off in response to signals generated by the patient's ventilatory drive (eg, changes in airway pressure caused by inspiratory and expiratory efforts).

End-expiratory pressure (EEP)—the baseline transrespiratory pressure that exists at the end of the expiratory time.

Event—any condition or occurrence related to mechanical ventilation that requires clinician awareness or action. *Technical events* are those involving an inadvertent change in the ventilator's performance; *Patient events* are those involving a change in the patient's clinical status.

External compressor—a device external to the ventilator used to supply pneumatic source power.

Expiratory phase (expiration)—the part of the ventilatory cycle from the beginning of expiratory flow to the beginning of inspiratory flow.

Expiratory time—the duration of the expiratory phase.

Gauge pressure—pressure at some point in space measured relative to atmospheric pressure. Positive gauge pressure is above atmospheric pressure, and negative gauge pressure is below.

Inspiratory phase (inspiration)—the part of the ventilatory cycle from the beginning of inspiratory flow to the beginning of expiratory flow. Any inspiratory pause is included in the inspiratory phase.

Inspiratory time—the duration of the inspiratory phase.

Internal compressor—a device inside the ventilator used to convert either pneumatic or electric source power into inspiratory pressure.

Limit—*verb*, to set a maximum value for pressure, volume, or flow during mechanically supported inspiration (or expiration); *noun*, the preset maximum value for pressure, volume, or flow during an assisted inspiration (or expiration). Inspiration (or expiration) does not terminate because the limit value has been met.

Mandatory breath—an assisted breath that is either initiated or terminated by the ventilator rather than by the patient's physiology or ventilatory drive.

Mean airway pressure—the average pressure that exists at the airway opening over the ventilatory period. It is usually measured as gauge pressure. Mean airway pressure is mathematically equivalent to the area under the time-pressure curve (from the beginning of one breath to the beginning of the next breath) divided by the ventilatory period.

Open-loop control—a control scheme in which the output of a system is determined by the initial setting of the controller with no corrections made to accommodate disturbances in the output caused by external factors.

Passive expiration—expiration not assisted by the ventilatory muscles or by any ventilator-induced change in transrespiratory pressure causing flow in the expiratory direction.

Phase—one of four significant events that occur during a ventilatory cycle: (1) the change from expiratory time to inspiratory time, (2) inspiratory time, (3) the change from inspiratory time to expiratory time, and (4) expiratory time.

Phase variable—a variable (ie, pressure, volume, flow, or time) that is measured and used to initiate some phase of the ventilatory cycle.

Phase variable value—the magnitude of a phase variable.

Spontaneous breath—an assisted breath that is both patient initiated and patient terminated; an unassisted breath.

Transient expiratory assist pressure (TEAP)—a mechanically created, transient baseline pressure that is lower than the end-expiratory pressure of the previous breath. This transient baseline lasts until the volume inspired during the previous breath has been exhaled, at which time the baseline pressure is increased to the previous end-expiratory pressure.

Transrespiratory pressure—the pressure difference between airway and body surface (ie, airway pressure – body surface pressure).

Trigger—*verb*, to initiate the inspiratory phase of an assisted breath.

Ventilatory period—the time from the beginning of inspiratory flow of one breath to the beginning of inspiratory flow for the next breath (total cycle time); the sum of inspiratory time and expiratory time; the reciprocal of ventilatory frequency.

APPENDIX
Determining Ventilator Classification

To determine which criteria (Fig. A-1) are met by a given ventilator and, thus, how the ventilator is to be classified, you must

(1) choose values for test-load parameters and (2) define waveform deformation.

The appropriate test-load values have been suggested by the American National Standards Institute (ANSI).[1] Waveform deformation can be defined in terms of one or two characteristics rather than the entire waveform. For example, for a given ventilator one might decide that if the peak value of the volume waveform (ie, the tidal volume) and the inspiratory time vary by no more than 20% (and other criteria from Fig. A-1 are satisfied), the ventilator can be classified as a volume-controller. In a similar way, the mean inspiratory pressure (not mean airway pressure) can be used with the criteria from Figure A-1 to determine whether a ventilator is a pressure-controller.

Step 1. Make initial observations. Study the text and diagrams in the operator's manual until you understand how the ventilator directs gas throughout a ventilatory cycle. Note particularly which variables are measured and whether open- or closed-loop control is operative. Attach a simple test lung to the ventilator circuit and become familiar with controls.

Step 2. Formulate an hypothesis. For example, if you observe that the ventilator has a control knob marked Tidal Volume, you might postulate that the ventilator is a volume-controller. Note the criteria from Figure A-1, consult Figure A-2, and note that at least two load levels must be simulated. From initial observations (ie, is the ventilator intended for adults only?), modify the hypothesis to specify the range of volume and flow over which the ventilator will perform as a volume-controller.

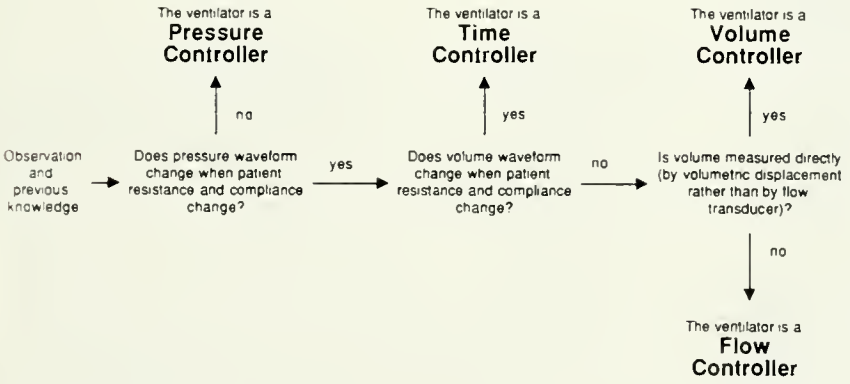
Observation and previous knowledge
Does pressure waveform change when patient resistance and compliance change?
yes
no
The ventilator is a Pressure Controller
Does volume waveform change when patient resistance and compliance change?
yes
no
The ventilator is a Time Controller
Is volume measured directly (by volumetric displacement rather than by flow transducer)?
yes
no
The ventilator is a Volume Controller
The ventilator is a Flow Controller

Fig. A-1. Criteria for determining the control variable during a ventilator-supported inspiration. Beginning with observations and previous knowledge of this ventilator, decisions are based on the effect of load on ventilator output.

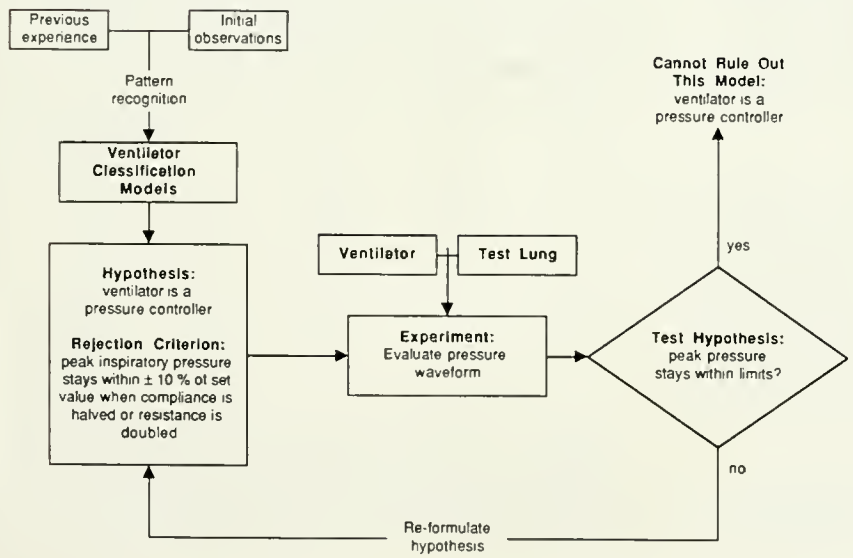
Previous experience
Initial observations
Pattern recognition
Ventilator Classification Models
Hypothesis: ventilator is a pressure controller
Rejection Criterion: peak inspiratory pressure stays within ± 10 % of set value when compliance is halved or resistance is doubled
Ventilator
Test Lung
Experiment: Evaluate pressure waveform
Test Hypothesis: peak pressure stays within limits?
yes
Cannot Rule Out This Model: ventilator is a pressure controller
no
Re-formulate hypothesis

Fig. A-2. The scientific deductive reasoning process applied to the evaluation of ventilator performance.

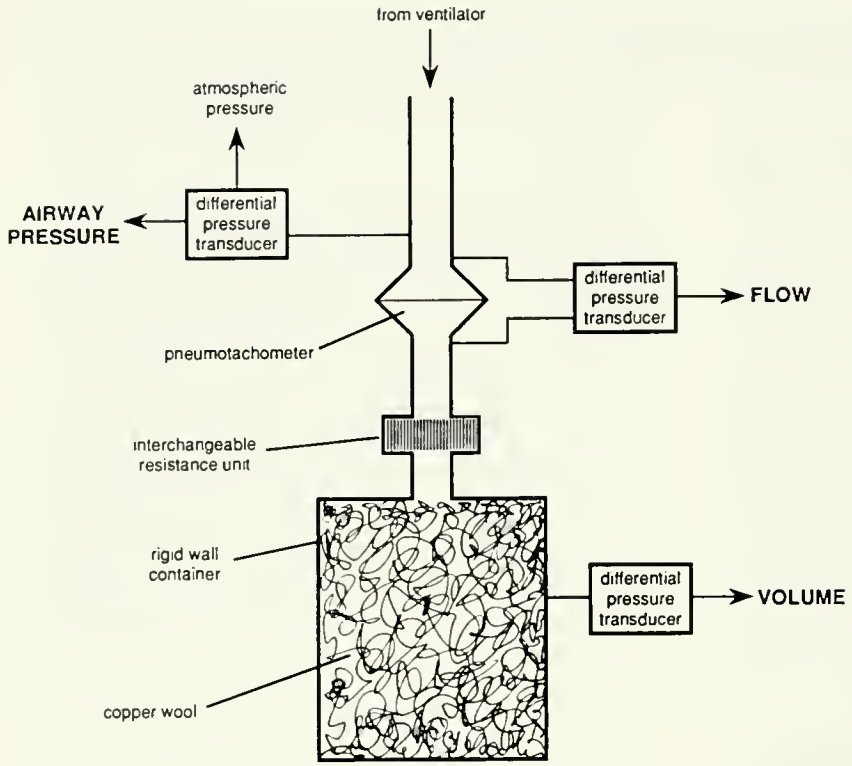

Fig. A-3. Experimental setup for evaluating ventilator performance characteristics with a passive lung model, according to ANSI recommendations.

Step 3. Define the rejection criteria. Also determine how much variation or what tolerance limits[2] are acceptable. If later it can be seen from a waveform recording that the waveform changes that occur as load changes exceed the acceptable limits, the hypothesis will be rejected.

Step 4. Design the experiment. Plan and make notes regarding (1) the measurements to be made, their sequence, and the number of repetitions, and the data forms to be used (a laboratory notebook can be useful here[3]); and (2) the number of units (ventilators of the same model) to be tested; (3) the lung model to be used. (ANSI[1] has described in detail containers for simulating respiratory compliance, and a discussion of the related calculations are available in the text by Chatburn and Craig.[4] Linear resistors can be constructed as described by Saklad and Weyerhaeuser.[5])

An experimental setup is shown in Figure A-3. Specifications for the required compliance and resistance are shown in Table A-1. During ventilator tests with the model (at frequencies up to 10 Hz), measurements of pressure, volume, and flow should be accurate within ± 2.5% of the reading.[2] Peak readings are allowed an additional tolerance of ± 2.5%. Power and work measurements should be accurate within ± 5% of the reading with an additional ± 5% tolerance for peak readings.

Step 5. Test the hypothesis. Compare the experimental data to the rejection criteria. Qualitative data is easily evaluated, but quantitative data from repeated measures must be summarized and expressed as mean values with standard deviations. From these basic statistics, tolerance limits[2] can be calculated. If the observed tolerance limits exceed the rejection-criteria limits, the hypothesis is rejected.

By following the five steps and referring to Figure A-4, phase variables can be determined.

Table A-1. Lung-Model Parameters Recommended by the American National Standards Institute for Evaluating Ventilator Performance[1]

Simulated Patient	Compliance (mL/cm H_2O)	Volume (L)*	Resistance (cm $H_2O \cdot s \cdot L^{-1}$)	Flow (L/s)†
Adult	50	51.6	5	0-2.0
	20	20.6	20	0-1.0
Child	20	20.6	20	0-1.0
	10	10.3	50	0-0.5
	3	3.09	200	0-0.1
Neonate	3	3.09	200	0-0.1
	1	1.03	500	0-0.075
			1000	0-0.05

* Volume = volume of rigid container required to achieved specified compliance when filled with 2% wire wool by volume at barometric pressure = 760 torr. See Reference 1.

† Range of flowrate for which resistance is linear.

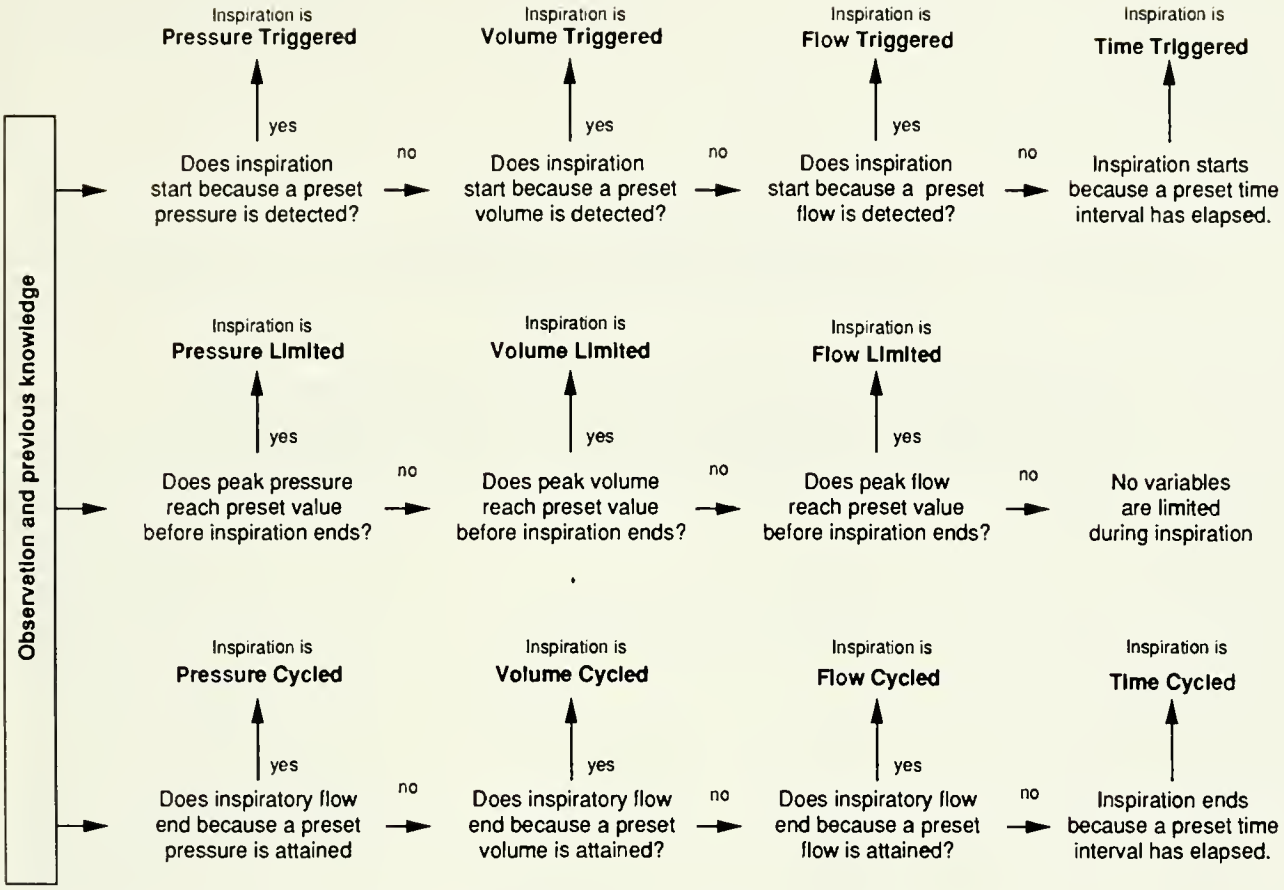
Observation and previous knowledge
Inspiration is Pressure Triggered
yes
Does inspiration start because a preset pressure is detected?
no
Inspiration is Volume Triggered
yes
Does inspiration start because a preset volume is detected?
no
Inspiration is Flow Triggered
yes
Does inspiration start because a preset flow is detected?
no
Inspiration is Time Triggered
Inspiration starts because a preset time interval has elapsed.
Inspiration is Pressure Limited
yes
Does peak pressure reach preset value before inspiration ends?
no
Inspiration is Volume Limited
yes
Does peak volume reach preset value before inspiration ends?
no
Inspiration is Flow Limited
yes
Does peak flow reach preset value before inspiration ends?
no
No variables are limited during inspiration
Inspiration is Pressure Cycled
yes
Does inspiratory flow end because a preset pressure is attained
no
Inspiration is Volume Cycled
yes
Does inspiratory flow end because a preset volume is attained?
no
Inspiration is Flow Cycled
yes
Does inspiratory flow end because a preset flow is attained?
no
Inspiration is Time Cycled
Inspiration ends because a preset time interval has elapsed.

Fig. A-4. Criteria for determining the phase variables during a ventilator-supported breath.

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5. Saklad M, Weyerhaeuser R. The construction of linear resistances for the testing of ventilators. Anesthesiology 1980;52:71-73.

CRCE through the Journal

For your information, answers to the 50 questions for ***CRCE through the Journal***, which appeared in the July issue of RESPIRATORY CARE, are given below. No scores will be available from the AARC until 1991 CRCE transcripts are released in early 1992. Deadline for submission of Answer Sheets for CRCE credit was September 3, 1991.

The correct answers to the questions are shown below.

1. b	14. e	27. b	40. d
2. a	15. b	28. d	41. c
3. d	16. d	29. c	42. b
4. b	17. d	30. e	43. a
5. b	18. e	31. d	44. e
6. a	19. a	32. b	45. a
7. b	20. d	33. c	46. b
8. d	21. e	34. no correct answer	47. e
9. d	22. c	35. a	48. d
10. c	23. c	36. a	49. e
11. c	24. c	37. a	50. b
12. e	25. a	38. c	
13. a	26. b	39. e	

Because of a production error, this test will be scored on the basis of 49 questions. (As written, there was no correct answer to Question 34.)

PFT Corner #41— Helium Flow-Volume Loop in an Asthmatic Patient

Robert Pickering MD and Fernando J Martinez MD

A 57-year-old man, a nonsmoker with a history of severe steroid-dependent asthma, nasal polyps, and aspirin allergy, was seen because of progressive breathlessness during the preceding year. Breathlessness had been present despite increasing doses of oral corticosteroids, inhaled steroids, inhaled albuterol, and cromolyn. His wife had noted his "noisy" breathing, particularly at night. Past medical history included laser excision of a vocal cord papilloma 2 years earlier.

Physical examination revealed normal breath sounds posteriorly but coarse breath sounds over the tracheal region. Results of routine laboratory studies were unremarkable. Radiography of the chest revealed normal lung fields and normal tracheal shadow. Results of spirometry with the patient first breathing room air and then an 80% helium-20% oxygen mixture (heliox) are shown in Table 1, and flow-volume loops are shown in Figure 1.

Table 1. Results of Spirometry in Asthmatic Patient Presenting with Increased Breathlessness and Noisy Breathing

	Room Air (% Predicted)	Heliox (% Predicted)	Room Air After Operation (% Predicted)
FEV_1 (L)	2.45 (71)	3.23 (80) 13*	3.31 (82)
FVC (L)	4.30 (84)	4.94 (97) 15*	4.99 (98)
FEV_1/FVC	66	65	66
PEFR (L/s)	3.88 (39)	4.70 (48) 26*	7.33 (76)
FEF_{50}/FIF_{50}	0.72	1.03	0.41
FIF_{50} (L/min)	231.60	161.40	97.00
$FEV_1/PEFR$ ($mL \cdot min \cdot L^{-1}$)	12.43	11.48	7.52
MVV/FEV_1	26.67		

*Percent improvement, heliox vs room air.

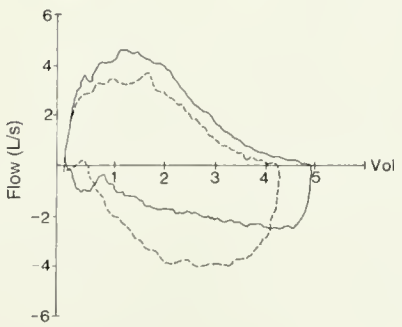

Fig. 1. Flow-volume loop performed on admission by patient breathing room air and a helium-oxygen mixture.

Questions

1. How do you interpret the spirometry and flow-volume loop?

2. What additional information does the helium-oxygen loop add?

3. What further studies are indicated?

Answers and Discussion on Next Page

Dr Pickering and Dr Martinez are associated with the Section of Pulmonary Medicine, Lahey Clinic Medical Center, Burlington MA.

Reprints: Fernando J Martinez MD, Section of Pulmonary Medicine, Lahey Clinic Medical Center, 41 Mall Road, Burlington, Massachusetts 01805.

1. Interpretation of Spirometry and Flow-Volume Loops: The decrease in FEV_1 confirms the presence of airflow obstruction. The suggestion of flattening in the expiratory limb of the flow-volume loop could represent poor effort or, possibly, variable intrathoracic airflow obstruction.[1]

2. Helium-Oxygen Loop Information: An increase in peak expiratory flowrate (PEFR) with maximal expiratory maneuvers performed while breathing a helium-oxygen mixture has been reported by some clinicians[2,3] to be useful in detecting upper-airway obstruction superimposed on coincident chronic airflow obstruction. In our patient, the 25% improvement in PEFR with heliox breathing (Table 1) suggests possible upper-airway obstruction.

3. Further Studies: Because of the clinical history of worsening asthma and spirometric suggestion of upper-airway obstruction, further study of the upper airway was indicated in this patient. Tracheal tomography results were normal, but fiberoptic bronchoscopy revealed extensive intrathoracic tracheal papillomatosis with high-grade obstruction of the tracheal lumen (Fig. 2).

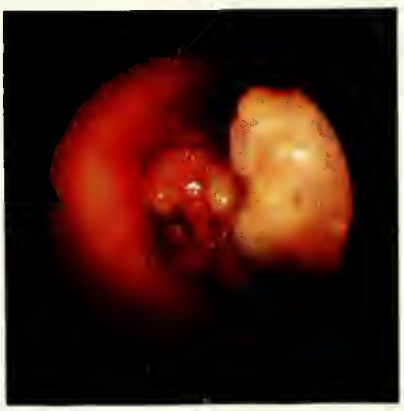

Fig. 2. Extensive papilloma almost completely obstructing the trachea.

Discussion

Upper-airway obstruction is known to masquerade as asthma[1,4,5] and also to be masked by worsening respiratory symptoms in patients with chronic airflow obstruction.[6,7] Miller and Hyatt[8] popularized the analysis of flow-volume loops in the diagnosis and characterization of upper-airway obstruction noting that flattening of the expiratory, inspiratory, or both limbs of the flow-volume loop permitted differentiation into variable intrathoracic, variable extrathoracic, or fixed upper-airway obstruction, respectively. This differentiation has recently been summarized by Acres and Kryger.[1]

A diagnosis of upper-airway obstruction can be tendered from examination of the flows on routine spirometry. Rotman and associates[9] suggest that $FEF_{50}/FIF_{50} > 1$, $FIF_{50} < 100$ L/min, $FEV_1/PEFR > 10$ mL · min · L^{-1}, and $FEV_1/FEV_{0.5} > 1.5$ may be used in this way. Owens and Murphy[10] added the calculation of $MVV/FEV_1 < 25$ and $FEF_{25-75\%}/FEF_{25-75\%} < 1$ as useful indicators of upper-airway obstruction as well. In our patient, only the FEV_1/PEFR of 12.43 was suggestive (Table 1). However, the clinical history of refractory asthma despite aggressive medical management, 'noisy breathing,' and the detection of possible stridor on physical examination raised suspicion of occult upper-airway obstruction.

The difficulty in detecting upper-airway obstruction superimposed on chronic airflow obstruction has been noted.[1] In assessing this point, one can take advantage of the fact that turbulence is directly related to gas density—the greater the density the greater the turbulence at a given flowrate. In the presence of upper-airway obstruction, turbulence is minimized and flow maximized when a gas of lower density is breathed.[11] Some authors[2,3] have noted that an increase in PEFR or maximum inspiratory flow ($\dot{V}I_{max}$) when a subject breathes a helium-oxygen mixture suggests upper-airway obstruction and can be used to detect such obstruction superimposed on chronic airflow obstruction. Although we noted a 25% improvement in PEFR while our patient was breathing heliox (Fig. 1), in the loop shown, the inspiratory flow appears to decrease when the patient breathes the heliox. This is an example of the marked variability noted in the inspiratory limb of the flow-volume loop with each maneuver. This may represent variable effort or the variability encountered in the performance of and interpretation of spirometry with helium-oxygen mixtures. This latter variability is one of the major difficulties encountered in the routine use of this technique.[12,13] Strict adherence to recommended techniques is obviously important.[14]

Data published by Despas and colleagues[15] and Antic and Macklem,[16] who examined heliox flowrates in stable patients with asthma, showed that in some asthmatic patients (particularly nonsmokers with no history of recurrent respiratory infection or chronic bronchitis), flowrates may increase when they breathe this lower density gas, suggesting a more central site for airflow obstruction. In our patient, the consistent increase in expiratory flow with heliox supported our decision to search for occult upper airway obstruction as the cause of the worsening symptoms. Bronchoscopy (Fig. 2) revealed greater than 90% obstruction of the distal trachea by a papilloma. After laser excision of the lesion, the symptoms improved greatly, as did the flowrate (Table 1) and flow-volume loops (Fig. 3). Therefore, we believe that the heliox flow-volume loops may be particularly useful in detecting occult upper-airway obstruction in patients whose symptoms worsen despite appropriate and aggressive medical management. Understanding the limitations of the technique is important in optimizing the information gained.

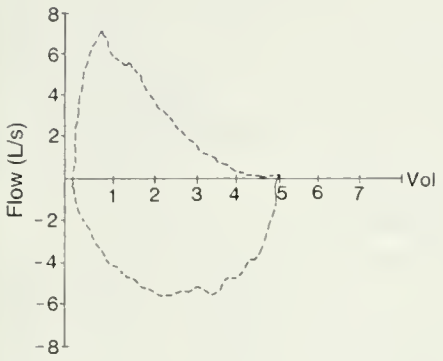

Fig. 3. Flow-volume loop performed by patient breathing room air after laser excision of papilloma shown in Figure 2.

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Tobacco Use in 1771, 1799, and 1957

I. A 1771 Encyclopaedia Article

Extracted from the article on Nicotiana in the First Edition of the Encyclopaedia Britannica.

The nicotiana tabacum, or tobacco, was firft brought into Europe about the year 1560, from the iſland Tobago in America. Beſides the tobacco of the Weſt Indies, there are conſiderable quantities cultivated in the Levant, the coaſts of Greece and the Archipelago, the iſland of Malta and Italy. Tobacco is either taken by way of fnuff, as a ſternutatory; as or a maſticatory, by chewing it in the mouth; or by ſmoking it in a pipe. It is ſometimes alſo taken in little longiſh pellets put up the noſe, where it is found to produce very good effects, to attract a deal of water, or pituita, unload the head, reſolve catarrhs, and make a free reſpiration; for the ſubtile parts of the tobacco in inſpiration, are carried into the trachea and lungs, where they looſen the peccant humours adhering thereto, and promote expectoration. Some have left this tobacco in their noſes all night; but this is found to occaſion vomiting the next morning. Another thing charged on this way of application, is, that it weakens the ſight. When taken in great quantities in the way of ſnuff, it is found to prejudice the ſmelling, greatly diminiſhes the appetite, and in time gives riſe to a phthiſis. That taken in the way of ſmoke, dries and damages the brain. Borrhi, in a letter to Bartholine, mentions a perſon who through exceſs of ſmoking had dried his brain to that degree, that after his death there was nothing found in his ſkull but a little black lump, conſiſting of mere membranes.

Some people uſe the infuſion of tobacco as an emetic; but it is a very dangerous and unjuſtifiable practice, and often produces violent vomiting, ſickneſs, and ſtupidity.

Bates and Fuller give ſome receipts, in which tobacco is an ingredient, with mighty encomiums in aſthma caſes. A ſtrong decoction of tobacco, with proper carminatives and cathartics, given clyſter-wiſe, ſometimes proves of good effect in what is uſually called the ſtone-cholic, and alſo in the iliac paſſion. A drop or two of the chymical oil of tobacco, being put on the tongue of a cat, produces violent convulſions, and death itſelf in the ſpace of a minute; yet the ſame oil uſed in lint, and applied to the teeth, has been of ſervice in the tooth-ach: though it muſt be to thoſe that have been uſed to the taking of tobacco; otherwiſe, great ſickneſs, reachings, vomiting, etc. happen; and even in no caſe is the internal uſe of it warranted by ordinary practice.

A ſtrong decoction of the ſtalks, with ſharp-pointed dock and alum, is ſaid to be of good ſervice, uſed externally, in cutaneous diſtempers, eſpecially the itch: ſome boil them for that purpoſe in urine. The ſame is ſaid to be infallible in curing the mange in dogs.

Beat into a maſh with vinegar, or brandy, it has been found ſerviceable for removing hard tumours of the hypochondria.

II. A 1799 Letter about Tobacco

Dated December 7, 1799, and printed on Page 1045 of Volume LXIX of ***The Gentleman's Magazine****, published in London, England*

Profeſſor Martin's remark on the cultivation of that remarkable herb, tobacco, has my teſtimony added to his own. It grows luxuriant in open grounds, and in waſte uncultivated lands of inferior ſoil, and even among rubbiſh, where few other plants would thrive. I think . . . it is worthy the notice of the Legiſlature in particular; for ſince America is no longer a province of ours, Government as well as the community would in time, by its cultivation here, derive advantage. It would employ our poor, and pay them well for their labour. It would lead to the improvement of our iſland, and would ultimately be a national profit. In many parts of Scotland as well as England it would anſwer; for its growth is quick, and, as it is dried in the ſhade, a wet autumn

would not injure its quality, though it might caufe lefs trouble to the planter in a dry one. I think there were many acres of tobacco cultivated in Roxboroughfhire fome years ago. Was it from a notion that the Englifh law prohibited its cultivation here? Or did a fubfequent act pafs including Scotland? The old acts of parliament I have feen on that head, seem to permit 200 plants, or half a fquare pole of land, in our gardens, for medicinal purpofes. What has been done lately I know not. It is a moft ufeful and valuable herb, and very falutary ufed moderately; and I never have heard that the inordinate or lavifh ufe of it did harm. I faw a gentleman within this month in the Northern part of our ifland, of very large fortune, very confiderable age, and, what is better, of very great worth, and refpected by all who know him, who told me he fmoked fometimes, being ufed to rife early, 10 or 12 pipes before breakfast for months in the winter; and a finer perfon at 70 you will rarely fee. There is a vulgar error gone forth, that it hurts the lungs. For myfelf, and fome of my friends, I can fay, that it does great good in coughs and afthmatic complaints, and has cured me of a diforder there. It is alfo faid, by difcharging phlegm and expectoration, to keep the body thin. It may, for anything I know; but the opinion of my friends, and the primary teftimony of my jacket, Sir, bear witnefs to the contrary. There is a pamphlet I have feen many years ago, when I abhored a pipe as much as I now love it; and have in vain hitherto of late enquired after it. The author is a clergyman; and it was written, I dare venture to fay, 40 years ago. I then thought the author fpoke too much for its medicinal qualities: now I incline to alter my opinion. It was, I think, intuited, "A Treatife on the Ufe and Abufe of Tobacco," and notices, in the Preface or Introduction, the cafe of a Queen or Princefs de Medici, who was cured of an old habitual cough by tobacco. I would be glad that you, or any of your smoaking friends, would direct me to it through your wide-extending Mifcellany.

Nicot.

III. Some 1957 Advice on Smoking during Pregnancy

Extracted from the book ***Expectant Motherhood****, Third Edition, Revised, by Nicholson J Eastman MD, published in 1957 by Little, Brown and Company, Boston. This item was discovered and submitted by Ken Moore RRT MEd, of Walla, Walla, Washington.*

Smoking.—A few years ago, Dr. Alexander M. Campbell of Grand Rapids, Michigan, sent a questionnaire to leading obstetricians in the United States asking the question: In your opinion does the smoking and inhalation of twenty-five or more cigarettes daily have an unfavorable effect on maternal health? Seventy-five replies were returned. Sixty-three, or 84 per cent, answered the question with "yes." Two, or 2.6 per cent, answered the question with "no." The ten others, that is 13.3 per cent, made replies which indicated some uncertainty. A number of obstetricians who answered "yes" qualified their replies by saying that moderate smoking is harmless.

While most obstetrical authorities, then, disapprove of excessive smoking in pregnancy (twenty-five or more cigarettes daily) there is no reason for believing that a woman who smokes moderately, let us say ten cigarettes or less a day, need change her custom at this time. If you have been used to smoking considerably more than this for several years, by no means try to give them up in pregnancy. There is no surer way of upsetting the nerves at a period when you should be calm or happy, or of converting a placid, sweet-tempered girl into an intolerable shrew. With negligible effort, even the most inveterate smoker can usually be content with a package a day or somewhat less, and if you arrange this there is no great cause for concern.

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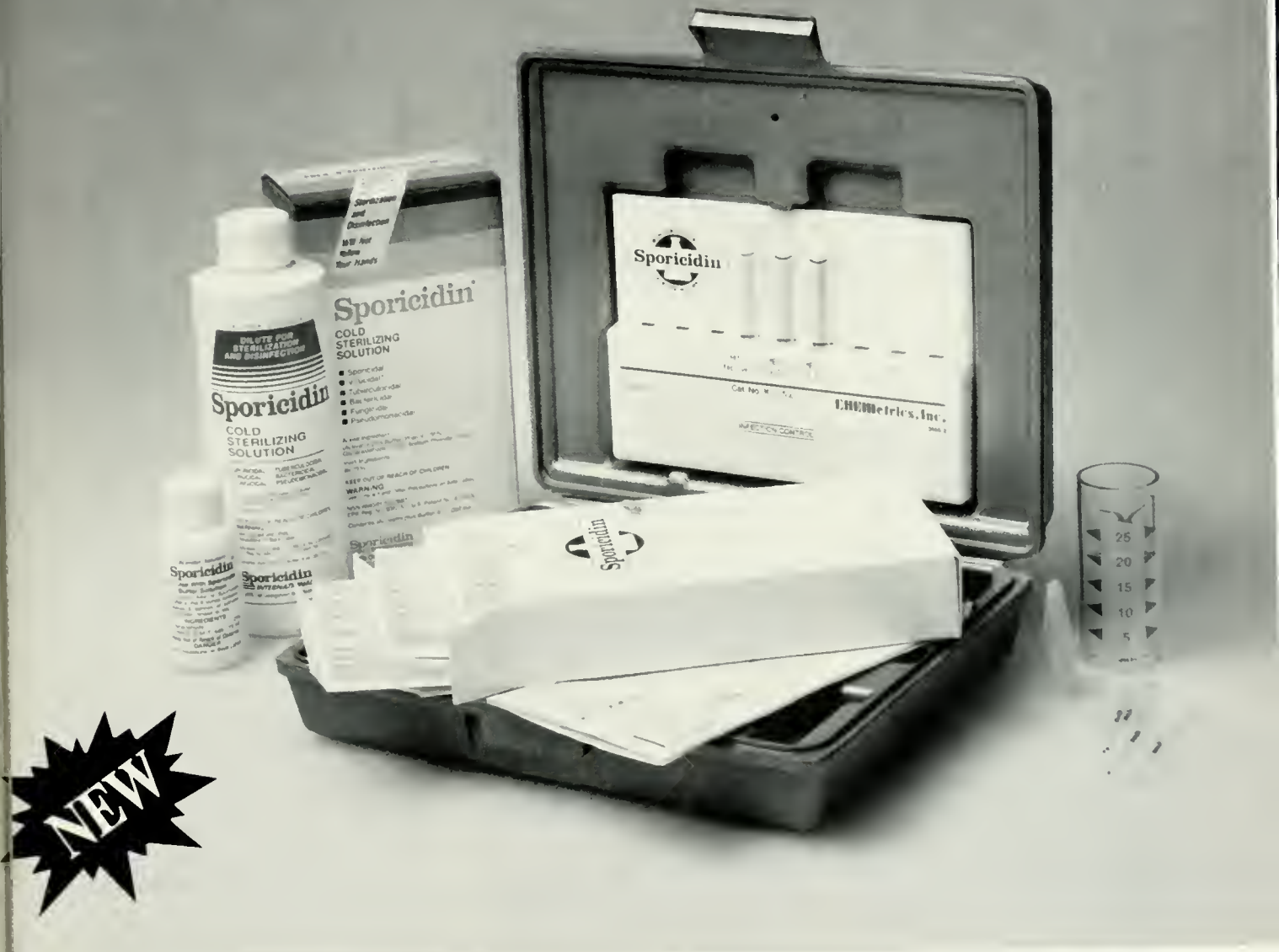

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Handbook of Neonatal Intensive Care, 3rd ed, by Henry L Halliday, Garth McClure, and Mark Reid. Hardcover, 416 pages, illustrated. Philadelphia: WB Saunders Co, 1989. $19.95.

The third edition of **Handbook of Neonatal Intensive Care** provides neonatal nursery clinicians with a convenient pocket reference text. The intent of the book is to enhance sound medical care by providing a referenced text that can be kept in one's pocket. The text provides details on the treatment of a variety of diseases encountered in the neonatal intensive-care setting.

For example, Chapter 4 deals with asphyxia and resuscitation; Chapter 6 highlights care of the low-birthweight infant, and Chapter 14 describes the processes of fetal and neonatal infection. Each chapter contains various charts and tables that enable one to quickly organize an approach to the treatment of a given disease process. The material presented is factual and succinct; the authors have simply highlighted modes of therapy and medical management.

Often, in the clinical setting, one only has time to take in the essentials of care and not delve into the esoterics of medical management. Each chapter is well referenced; therefore, one can, when convenient or necessary, check the reference listings and pursue topics in an exhaustive manner. Another interesting and helpful section of this text is the segment on procedures; this portion of the book explains procedures ranging from bag-mask ventilation to pericardiocentesis.

The authors have annotated a great deal of procedural information and placed it into a concise format. Procedures described in the text, as well as the suggested modes of treatment for various disease processes, are purposely abridged. This is not a shelf textbook; it is a clinician's guide to the care and treatment of neonatal patients. Appendices are also included that cover topics such as the Siggaard-Andersen nomogram, a description of the Bourns BP200 operating circuit, drug dosages, growth charts, and normal blood chemistry values.

The primary readership for this text is nurses and junior medical staff; however, I believe there is a wealth of information to be garnered by the respiratory care practitioner.

Chapter 10, "Respiratory Problems," is similar to the other chapters. A very brief and introductory treatment is given to ventilation philosophies and modalities; however, simply reading these guidelines and suggested modes of treatment in the respiratory problem section does not qualify one as a respiratory care practitioner. Neither does one qualify as a gastroenterologist by reading Chapter 18, "Gastrointestinal Problems." However, after reading Chapter 10, the gastroenterologist can understand a few of the basic concepts of mechanical ventilation; and after reading Chapter 18, the respiratory care practitioner can develop an appreciation of gastroenterology—thus both may be better practitioners. That is to say, they both will have a better picture of the neonate as a whole rather than as just a set of lungs or a necrotized loop of bowel.

The authors have fulfilled their intent and have done an excellent job of condensing a great deal of information into a portable text. The material presented is easy to follow and well organized. I believe this handbook will be helpful to both the experienced practitioner and the inexperienced—including respiratory care clinicians, nurses, and interns.

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Handbook of Respiratory Care, edited by Robert L Chatburn RRT and Marvin D Lough RRT. Softcover, 467 pages, illustrated. St Louis: Mosby Year Book Inc, 1990. $21.95.

The knowledge and information that a respiratory care practitioner needs to adequately assess and care for patients is astonishing—knowledge that not only includes what we know, but more importantly *knowing how to know*. An integral part of knowing how to know is knowing where to find information; it is in that spirit that **Handbook of Respiratory Care** is written.

Handbook of Respiratory Care condenses an enormous amount of clinically relevant data into a small, easy-to-use, reference guide. The book is organized into three main categories: patient assessment, therapeutics, and general science. It is written in outline form using boldface lettering for main topics and italicized lettering for sub-topics, making the search for specific data quick and the reading easy. Tables and figures are used frequently and appropriately to enhance the reader's understanding of the material. Numerous formulas and equations are used throughout the book—which at times may cause the reader to gasp for a breath of fresh air, but they are the meat of the text and make it the most comprehensive resource handbook available. After all, that is the editors' intent; it was

not written in an attempt to entertain you with light reading during break time or between treatments!

Chapter 1, authored by Dean Hess, defines the terms and techniques used in the "Physical and Radiologic Assessment of the Respiratory Patient." This chapter is well organized and complete, using many charts and figures to convey information; however, the radiologic assessment section could be improved with the addition of some abnormal chest x-rays (normal P-A and lateral films are illustrated). A reference list is included at the end of the chapter for readers seeking more detailed information.

Chapter 2, "Pulmonary Functions," includes a basic definition of tests and terms that are common to a pulmonary function laboratory, and has been upgraded to include information on exercise physiology. Normal values and prediction equations are included, while the clinically accepted range and significance of each is left to the practitioner and/or institution. A reference list is also provided at the end of this chapter for suggested reading, but the most current text in this list is dated 12 years ago.

Chapter 3, "Physiologic Monitoring," is likely the most clinically relevant chapter in the book. Equations and nomograms aid in the definition of various gas exchange relationships and in the analysis of arterial blood. Hemodynamic and electrocardiographic monitoring are also discussed in detail, and many excellent diagrams and waveforms are used to complement the text.

Chapter 4 is entitled "Gas Therapy," and explains all clinically pertinent gas laws, gas cylinder specifications and duration calculations, and oxygen administration. A reference list is provided for suggested reading.

A definite highlight of this handbook is Chapter 5 "Mechanical Ventilation." This chapter is divided into three sections: resuscitation equipment, definition of terms, and classification and operational characteristics of mechanical ventilators. The first two sections are detailed and complete with standard abbreviations and formulas. The last half of Chapter 5 is dedicated to the classification of mechanical ventilators (which is new and complex), but builds from simple fundamentals in an easy-to-follow, consistent, and understandable fashion. After reading this chapter, the practitioner will not only better understand the mechanical function of ventilators, but will be able to predict and describe how the ventilator will interact with the patient. Once accepted, this classification schematic will obviate the need and use of "catch phrases," "ventilator lingo," and inconsistencies in the medical community. The classification actually describes the type (mode) of ventilation used—just as pressure-support ventilation is referred to as patient-triggered, pressure-limited, flow-cycled ventilation; airway pressure release ventilation (APRV) is called time- or patient-triggered, pressure-limited, time-cycled ventilation, allowing spontaneous ventilation throughout the total cycle time. Tables comparing the operational characteristics of many commonly used neonatal and adult ventilators are also included.

Chapter 6 is the Formulary, which not only includes definitions of pharmacologic terminology and drug dosage calculations but also contains an exhaustive list (over 125) of modern-day drugs utilized in the intensive care of patients with respiratory disorders. The list is not limited to respiratory drugs alone but also includes antibiotics, sedatives, cardiac, and vasoactive drugs. Dosage, route of administration, action, and side effects are listed for each drug. Chapter 7 consists of 10 tables summarizing normal hematology and chemistry values.

Chapter 8 is Chatburn's "refresher course" on mathematics. Knowledge and understanding of mathematical procedures seems to be a very weak area for most respiratory care practitioners. Without that understanding, many of the formulas and equations punctuating the pages of this handbook have limited use. A section on statistical procedures and terminology not only aids researchers but clinicians trying to read and understand their work. A newly added section of this chapter defines and provides conversion factors for SI Units (International Systeme de Unite) used to record numerical values.

The handbook is complete with a 42-page Appendix that provides numerous medical abbreviations and definitions, translation of commonly used medical terms to five different languages, tables containing miscellaneous reference data, and illustrations displaying appropriate positions for postural drainage.

Chatburn and Lough are to be commended for their interest and efforts in providing such an extensive amount of data in a small, easy-to-use, and affordable handbook. **Handbook of Respiratory Care** provides information essential for the care of pediatric and adult patients with respiratory disease. I believe that when such information is in the hands of the health-care professionals providing patient care (physicians, therapists, nurses, and students), care will improve.

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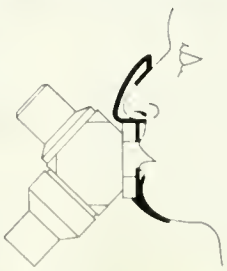

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Letters

Comments on Portable Airway-Suction Systems

We read with interest the study entitled, "Portable Airway-Suction Systems: A Comparison of Performance."[1] Curran and his colleagues raise an interesting question about the appropriateness of the recommendation that a device be capable of producing a minimum of 300-torr suction pressure, a recommendation that has been endorsed by the American Heart Association and utilized by various testing organizations and state authorities despite little data to support its value.[2-4] Although adequate suction pressure is one component to consider in the evaluation of an effective device, high pressures alone do not determine its ultimate performance. Furthermore, Curran and his colleagues point out that pressures much lower than 300 torr are recommended to avoid tracheal injury.[5,6]

We recently completed a comparative study of prehospital, portable suction devices utilizing a methodology that combines input from a user survey and a field-use simulation (simulated oropharyngeal secretions). When these data are compared to standards such as suction pressure > 300 torr, only weak correlation is seen. In other words, the ability of a device to perform with simulated oropharyngeal secretions correlates poorly with its ability to produce high pressures.[7]

With regard to the more practical, comparative evaluation, Curran et al chose imitation maple syrup because of its consistent, high viscosity. Although this may simulate the nature of thick mucus, it bears little resemblance to the more troublesome oropharyngeal substance, gastric emesis. Our experiences suggest that clogging, due to the particulate nature of emesis, is the greatest challenge facing portable suction devices. This point is well illustrated by the fact that when Curran et al attempted to use chunky baby food in their simulation, clogging of the 14-Fr Yankauer tube was a continual problem. Rather than abandoning the more realistic, nonhomogeneous medium (baby food), we suggest omitting the use of the Yankauer tube. This is a technique well known to paramedics who use these devices daily for suctioning gastric emesis.

In summary, we share Curran and his colleagues' concern that the minimum standard of 300-torr pressure may be inappropriate. Furthermore, there is evidence to support the concept of limiting maximum pressure of such devices to avoid tracheal injury. In addition, we feel that standards for these devices must include performance criteria that more accurately reflect the conditions under which these devices are used.

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Matrx Suction System Offers Optional Regulator

In the article "Portable Airway-Suction Systems: A Comparison of Performance" (Respir Care 1991;36: 259-266), I believe that the authors make an inaccurate statement. Matrx has offered an optional regulator/gauge since 1986. The vacuum regulator accessory allows the operator to vary suction from 0 to –550 torr (maximum vacuum). The regulator consists of a suction control valve, gauge, tee fitting, and silicone tubing that can be added to or deleted from Matrx/Ohmeda suction units. The part number is 92120129. Readers are

welcome to call me for additional information at (800) 847-1000.

Gary E Kaczor
Vice President of Marketing
Matrx Medical Inc
Orchard Park, New York

The authors respond:

We thank Dr DiLorenzo and Dr Stewart for voicing their concerns and observations so clearly—and we agree completely.

We also thank Mr Kaczor for the information on the Matrx/Ohmeda pressure-regulation option. As indicated in the article, we evaluated each instrument as manufactured, without options or attachments. We hope that users of the device will incorporate the attachment that he describes.

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More on Chest Physiotherapy

I wish to applaud Eid et al for their recent and valued paper, "Chest Physiotherapy in Review."[1] To be concise but detailed, which is the goal of a review, is no easy task. In 1985, Kirilloff et al, reviewing the then extant literature, concluded that CPT in general had not been shown to be useful, and that percussion, in particular, had not been demonstrably effective.[2]

The indications, techniques, and efficacy of CPT have preoccupied me for a number of years, and I write this letter with several objectives: (1) to add further evidence to support Eid et al's conclusions as to the selective value of CPT, (2) to address the benefits of percussion, (3) to speak to omissions in Eid et al's paper regarding the reported Britton et al 1985 study on pneumonia patients[3] and the Reisman et al 1988 study of cystic fibrosis patients,[4] and (4) to assume a prosecutorial tone and editorialize on the wrongfulness of some physicians' CPT-ordering practices. The goals of CPT are to improve mucociliary clearance, to increase the volume of sputum expectorated, and to improve airway function. The underlying presumption is that when these goals are accomplished, lung collapse will be prevented, minimized, or reversed; and that the removal of excess secretions will forestall the occurrence of chest infection. Lofty aims and logical premises do not legitimize therapy and techniques whose efficacy remains at least unsupported, if not disproved. Yet, the use of CPT prophylactically and therapeutically for all patients with a variety of anticipated and actual pulmonary problems has become well entrenched in the standard of medical care.

Intuitively and intellectually we know that CPT does not work for all patients. Writing to the *British Medical Journal* in 1989, a spokesperson for the Chartered Society of Physiotherapists bemoaned in a letter that, "Physiotherapists are aware that physiotherapy for all is not the answer."[5] Is there a reader who has not felt similarly exasperated? A 1990 editorial by Selsby in the same journal admonished physicians on the selectivity of indications and the variability of benefits.[6]

Shared by many physicians, nurses, patients, and therapists is the belief that CPT is percussion, and that, moreover, unless the rhythmical sound of clapping has been heard, a CPT treatment has not been administered. The persistence of percussion as a nearly universally applied technique by unquestioning therapists is both mystifying and distressing. Kirilloff et al reached the startling conclusion in a 1985 paper that no data existed to support the independent and beneficial effect of percussion or vibration.[2] Other reports published at about the same time supported that conclusion. In patients with chronic bronchitis and excessive secretions, the addition of percussion to postural drainage "made no significant difference" at the Brompton Hospital in England,[7] was of "little value" in a study from Sweden,[8] and improved secretion removal only "slightly" in a report from the Netherlands.[9]

Percussion has not merely been questioned; in the case of acute asthma it has been condemned. In a 1990 statement of guidelines for the management of asthma in adults, the British Thoracic Society wrote that "percussive physiotherapy is contraindicated."[10] Support for percussion is often postulated upon the studies of Bateman et al, who in 1979 reported CPT to be beneficial in sputum removal,[11] and who in 1981 wrote that CPT and cough were more effective than cough alone.[12] However, they made no attempt to distinguish the effects of percussion independently—and the CPT administered included postural drainage, vibration, shaking, and coughing. One of the authors of those two papers, Clarke, has since then (in a 1987 letter written with Sutton)[13] disavowed percussion in favor of the forced expiration technique (FET) with postural drainage.

Does percussion have any place in the treatment of patients? Gertrude Stein, author and Johns Hopkins medical school graduate, once said that "A difference to be a difference must make a difference." Van Der Schans et al, in their Netherlands study reported in 1986, did allow the theoretical possibility of benefit from percussion when patients were capable of effectively coughing and/or tolerating postural drainage.[9] Radford and others (1983) showed that, in the absence of coughing, mechanical percussion altered the character of a sputum bolus, causing it to spread longitudinally, hypothesizing that this

change increased its transport rate. They admitted, however, that the optimal percussive rate was, regrettably, beyond manual capabilities. At our facility, we consider percussion when other methods of secretion clearance are having no success; if a trial of percussion makes a difference (the Gertrude Stein effect), then it is incorporated into the treatment; if not, it is abandoned.

Eid et al correctly reported that in the Britton et al study of CPT in pneumonia patients[3] there were no significant differences between the study group (CPT) and the control group with respect to either duration of hospital time or healing time. It was their objective conclusion that CPT "is at best useless in patients with primary infectious pneumonia." Eid et al failed in their review to mention that virtually every patient in the Britton et al study who had received CPT returned a questionnaire that cited CPT as being of considerable benefit in the healing process. The physical presence several times daily of someone who is performing an evaluative, tactile, social service conjures up an image of attentiveness and intensity. For pneumonia, is this then the service we provide—a pretence? [pretense]

It is true that the Reisman et al prospective study of young patients with cystic fibrosis[4] did demonstrate a deterioration in pulmonary function over 3 years when the FET-only group was compared to those receiving FET plus conventional CPT, which included postural drainage, percussion, and vibration. However, these findings may be spurious because the FET was not done in association with postural drainage, as advocated by some,[15] which has been shown to improve pulmonary function more than does postural drainage alone.[16]

We must conclude that, despite clinical impressions to the contrary,[17] much of the CPT we administer is of dubious benefit. What is indisputable is that, as a modality, CPT is hardly a benign exercise. On the contrary, it is a stressful procedure that has the potential for serious metabolic, neurologic, and hemodynamic sequelae, including death.[18-21] There is also the impact on the caregiver. Performing unnecessary therapy is destructive to departmental morale and dispiriting to the individual.

It is not inconceivable that across the nation there are unquestioning therapists administering chest physiotherapy as often and as long as the physician has ordered. There is the underlying assumption that all physicians and residents know how to recognize those patients who would benefit by being treated. Experience and published studies are evidence to dispute this. Are these adverse side effects and complications then unnecessary? By what reasoning or right do physicians persist in ordering therapy that does not improve outcome? By what right do respiratory therapy departments charge patients for these interventions? Is it a matter of following orders? Is it fear of the legal consequences of not following orders? Where is the legal defense when a known possible adverse consequence is caused by the administration of therapy proven unworthy? More importantly, with respect to patients' rights, are they being informed that CPT is being administered without acceptable evidence that it will help them, or are they even being informed that there is no reasonable expectation that this therapy will improve the outcome for them? In the absence of clear-cut demonstrable benefit of CPT, the only acceptable rate of excess therapy should be zero.

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20. Mackenzie C. Ciesala N, Imle P, Klemic N. Chest physiotherapy in the intensive care unit. Baltimore: Williams & Wilkins, 1981:211-219.
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Dr Eid replies:

The reasons we wrote our paper about chest physiotherapy (CPT)[1] were to promote much needed research in the field and to further exchange of ideas about this modality of treatment. I am extremely pleased to see that Mr Barber has responded to this challenge.

Mr Barber's concern about the uses and abuses of CPT was the main driving force behind the two years of research that led to our paper. Being at both the receiving end of CPT orders (Medical Director of the Respiratory Care Department) and the ordering end (Director of Pediatric Pulmonary Service, which involves writing many orders about various forms of treatment, including CPT), I can appreciate the frustration and distress that Mr Barber feels.

I would like to reiterate here the findings stated in our paper: (1) CPT still has many indications, which we detailed; (2) knowledge about those indications is of paramount importance to both doctors and respiratory caregivers; and (3) communications between ordering physicians and respiratory caregivers should be maintained and discussion should be open and frank in order to deliver optimal medical care. We could not in our review discuss in detail specific treatment procedures, such as type of CPT, frequency, duration, and position. I am thankful to Mr Barber, who discussed in some detail the benefit of percussion. I concur completely with him about Reisman et al's study,[2] which omitted postural drainage and percussion from the FET group. Our omission regarding the Britton et al study[3] was deliberate, as we feel that returning a questionnaire 2 months after an acute illness is far from scientific.

It is distressing physically, economically, and medically to see so many inappropriate orders for CPT. However, we are a long way from Mr Barber's conclusion that "in the absence of clear-cut demonstrable benefits, the only acceptable rate of excess therapy should be zero." Although I concur with this strong statement, education about CPT, communication between physicians and respiratory caregivers, and exchange of ideas (such as in Mr Barber's letter and this reply) are needed for a better understanding of such a complex issue.

I hope such exchanges continue as we gain more knowledge, and that more controlled studies will be conducted to address the many unanswered questions raised in our paper and in Mr Barber's letter.

Nemr S Eid MD
Director
Pulmonary Medicine
Kosair Children's Hospital
University of Louisville
Louisville, Kentucky

REFERENCES

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2. Reisman JJ, Rivington-Law B, Corey M, Marcotte J, Wannamaker E, Harcourt D, et al. Role of conventional physiotherapy in cystic fibrosis. J Pediatr 1988;113:632-636.
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Not-for-profit organizations are offered a free advertisement of up to eight lines to appear, on a space-available basis, in Calendar of Events in RESPIRATORY CARE. Ads for other meetings are priced at $5.50 per line and require an insertion order. Deadline is the 20th of month two months preceding the month you wish the ad to run. Submit copy and insertion orders to: Calendar of Events, RESPIRATORY CARE, 11030 Ables Lane, Dallas TX 75229-4593.

Calendar of Events

AARC & AFFILIATES

October 15-17 in Atlantic City, New Jersey. The NJSRC presents the 1991 Shore Conference at Trump Castle by the Bay. Contact Gail Horn RRT, Kennedy Memorial Hospital-Cherry Hill, PO Box 5009, Cherry Hill NJ 08034. (609) 488-6847.

October 17-18 in Columbia, Missouri. The Missouri Society District III presents the 3rd Annual "Current Concepts in Respiratory Care." Topics include capnography, reimbursement issues in RC, current practices/alternatives for CPT, and transtracheal oxygen therapy. An open discussion is presented on euthanasia and assisted dying, featuring a hospital chaplain, physician, and professor of medical ethics. Contact Kelly Dockins RRT, Technical Director of Respiratory Care, AMI Columbia Regional Hospital at (314) 875-9296.

October 18 in Long Island, New York. The Southeastern Chapter of the NYSSRC presents its 23rd Annual Symposium, "Professional Practice Update 1991," at the Marriott Hotel in Uniondale. Speakers include Patrick Dunne MEd RRT (Continuous Quality Improvement); Marty Douglass MA (Clinical Applications of Noninvasive Ventilation); Barbara Finger JD (Clinical Incident Documentation); Thomas East PhD (The Use of Computers in Respiratory Care); Aimee Telsey MD (Artificial Surfactant Replacement); and Fred Mindermann BS RRT (Dynamic Interaction Ventilation). Contact Ken Axton RRT at (516) 444-3180.

October 25 in Bear Mountain, New York. The Hudson Valley Chapter of the NYSSRC presents its annual educational seminar at the Bear Mountain Inn. The theme for the event is "Pediatric Ventilation & Respiratory Care." The magnificent landscape, a buffet lunch, the scenic Hudson River, and fall foliage complement the event. Contact Mike Aiello RRT, Box 150, Glenham NY 12527. (914) 795-5340.

October 25 in San Diego, California. The University of California San Diego Medical Center, in collaboration with the CSRC (Chapter I), presents "Respiratory Update II." Seminar topics range from ventilator-patient pulmonary mechanics to advances in neonatal respiratory care. *Continuing education pending through CSRC.* Contact Rick Ford at UCSD Medical Center, H-771, 225 Dickinson St, San Diego CA 92103. (619) 543-2593.

October 25 in Spokane, Washington. The Eastern Chapter of the WSRC presents "Special Techniques: Taking the Respiratory Care Profession into the 21st Century." Featured special guests include Forrest Bird PhD; Dr Icenogle; and Jerry Focht RRT. Contact Dee Arkell RRT, Spokane Community College at (509) 536-7310 or (509) 924-1197.

October 30-31 in Sturbridge, Massachusetts. The MSRC presents its 14th Annual Meeting, at the Sheraton Sturbridge Resort and Conference Center. Topics include bronchoscopy, home care, asthma diagnosis and therapy, ECMO, and sleep studies. Special feature is a debate covering the pros and cons of pentamidine and ribavirin. Contact Gina Farquharson RRT, 16 Bartlett St, Pembroke MA 02359. (617) 293-6090.

November 15 in Cuyahoga Falls, Ohio. The OSRC's Homecare Committee hosts its first seminar, "Homecare in the Nineties." Topics include the impact of home care accreditation, designing a course for respiratory home care education, and pressure-supported ventilation in the home. Contact Robin Garland at (216) 744-9071.

OTHER MEETINGS

October 17-18 in Birmingham, Alabama. The Children's Hospital of Alabama presents its 1st Annual Perinatal-Pediatric Respiratory Care Clini-Course at the Radisson Hotel. Topics include assisted ventilation of the neonate, high-frequency ventilation, pressure-support ventilation, mechanical ventilation of the asthmatic patient, pediatric trauma, mask ventilation, delivery of aerosolized drugs, and continuous aerosolization. Speakers include Waldemar Carlo, Robert Kacmarek, Allen Goldberg, and Joseph Rau. Contact Susan Forrest at (205) 939-9675.

October 18 in Cincinnati, Ohio. The University of Cincinnati presents "Understanding Lung Function Tests." Contact Occupational Pulmonary Services at (513) 558-1234.

October 30 in Huron, Ohio. The American Lung Association of Ohio's South Shore and the Sandusky Area Health Education Center co-sponsor the 2nd Annual North Coast Pulmonary Disease Conference, "Current Modalities in Pulmonary Care," at the Sawmill Creek Resort Hotel. Contact Carol Adler at (419) 663-5864 or (800) 231-5864.

November 7-8 in Des Moines, Iowa. The respiratory care departments of Iowa Methodist Medical Center and Mercy Hospital Medical Center, in conjunction with the ISRC's fall meeting, sponsor the 2nd Annual Respiratory Seminar at the Mercy Education Center. Contact Delite Lester RRT, Education Coordinator, Mercy Hospital Medical Center at (515) 247-4197.

November 8-10 and February 14-16—Patient Cruise/ Floating Seminar. Cruise from Miami to Nassau. Rates include private stateroom (double occupancy), all on-board meals and entertainment, and prescribed respiratory care services. Four CRCEs awarded on "Clinical vs Physiologic Monitoring." Deadline is Oct 4 and Jan 3, respectively. Contact Dave Robbins at (305) 441-6819 or (305) 232-1908.

November 15 in Portland, Maine. Maine Medical Center, Division of Pulmonary Medicine, presents "Respiratory Care: A Look Back and To the Future," at the Dana Education Center at Maine Medical. The conference provides a comprehensive overview of the past, present, and future trends in respiratory diseases, neonatal care, respiratory technology, respiratory intensive care, surgical treatment for respiratory diseases, and medical ethics. Contact Tim Blanchette RRT at (207) 871-2662.

December 6 in Cincinnati, Ohio. The University of Cincinnati presents "Spirometry Refresher—Update." This course reviews recent changes in spirometry testing standards, guidelines, and interpretation techniques. Contact Occupational Pulmonary Services at (513) 558-1234.

January 20-27 in Bahamas Cruise/Orlando Tour. Dream Cruises' "Stress Busters Cruise" for continuing education combines lectures and workshops throughout the week. $795 (per person/double occupancy) inside cabin or $825 (per person/double occupancy) outside cabin. Price includes airfare, cabin, hotel, car, transfers, with food and entertainment provided aboard ship. Contact Kathy Kearney at (800) 462-3628, or write 10882 LaDona Ave, Garden Grove CA 92640.

February 12-14 in Mexico City, Mexico. The Consejo Mexicano de Inhaloterapia presents the 1st International Congress of Respiratory Therapy at the Hotel Paraiso Radisson Perisur. International speakers and exhibits are featured. Contact Dr Hector León Garza at 011-525-588-7386, or fax 011-525-578-8952.

For your convenience, information, and direct access, the advertisers in this issue and their phone numbers are listed below. Please use this directory for requesting written material or for any questions you may have.

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RESPIRATORY CARE

Instructions for Authors and Typists

These Instructions are meant to guide authors and typists, including veterans in those roles, in the production of quality manuscripts. Perfection is not expected, but the well-prepared manuscript has the best chance for prompt review and early publication.

General Requirements

Submissions should (1) be related to respiratory care, (2) be planned for one of the publication categories below, and (3) be prepared as indicated in these Instructions. A letter accompanying the manuscript must specify the intended publication category, be signed by all the authors, and, when there are two or more authors, state that "We, the undersigned, have all participated in the work reported, read the accompanying manuscript, and approved its submission for publication."

Publication Categories

Research Article (Study): A report of an original investigation.
Evaluation of a Device/Method/Technique: A description and evaluation of an old or new device, method, technique, or modification.
Case Report: A report of a clinical case that is uncommon or of exceptional teaching value. The author(s) must have been associated with the case. A case-managing physician must be one of the authors or, if not an author, must supply a letter approving the manuscript.
Case Series: Like a Case Report but including a number of cases.
Review Article: A comprehensive, critical review of the literature and state of the art of a pertinent topic that has been the subject of 40 or more published research papers.
Overview: A critical review of a pertinent topic about which not enough research has been published to merit a Review Article.
Update: A report of subsequent developments in a topic that has been critically reviewed (not necessarily in this journal).
Point of View: A paper expressing the author's personal opinions on a pertinent topic.
Special Article: If a paper does not fit one of the foregoing categories but is pertinent, the editors may consider it as a Special Article.
Editorial: A paper that draws attention to a pertinent concern.
Letter: A signed communication about material published in this journal or on topics of interest or value to readers.
Blood Gas Corner: A brief, instructive case report (real or fictional) involving invasively or noninvasively obtained respiratory care blood data, followed by questions for readers—with answers and discussion.
PFT Corner: Like Blood Gas Corner but involving pulmonary function testing.
Test Your Radiologic Skill: Like Blood Gas Corner and PFT Corner but involving pulmonary-medicine radiography and including one or two 4 × 5 or 5 × 7 inch prints of radiographs. The case must be real.
Review of Book, Film, Tape, or Software: Anyone interested in writing a review can discuss it with an editor.

Editorial Consultation and Author's & Typist's Kit

To discuss a writing project, write to RESPIRATORY CARE, 11030 Ables Lane, Dallas TX 75229 or call 214/243-2272.

Authors are urged to obtain the RESPIRATORY CARE Author's & Typist's Kit. The Kit provides *authors* with specific guidance about writing a research paper, writing a case report, converting to and from SI units, and in-house manuscript review. *Typists* can use the Kit's Model Manuscript, a list of journal name abbreviations, and a copy of these Instructions. The Kit is free from the Journal office.

Preparing the Manuscript

General Concerns—*Typist*

- Double-space ALL lines, including those in references, figure legends, and tables. Do not justify right margins.
- Number pages in upper right corner and leave margins of 1½″ or more on all four sides of the page.
- For research articles, follow format of Model Manuscript, Respir Care 1984;29:182 (Feb 1984).
- Meticulously follow instructions for typing references.

General Concerns—*Author:*

- Structure manuscript as specified hereafter.
- Provide all requested information on title page as specified hereafter.
- Proofread manuscript for completeness, clarity, grammar, spelling; be sure all references, figures, and tables are cited in the text.
- Consider having paper reviewed in-house before submission.
- Have all co-authors proofread and approve manuscript and sign submission letter.

Manuscript Structure

Most kinds of papers have standard parts in a standard order. However, papers can vary individually, and not every paper will have all the parts listed here.

Research Article: Title page, abstract page, continuous text (Introduction, Materials & Methods, Results, Discussion), Product Sources page, Acknowledgments page, references, tables, figure legends. Please consult "Writing a Research Paper," Respir Care 1985;30:1057 (Dec 1985) and Model Manuscript, Respir Care 1984;29:182 (Feb 1984).
Evaluation of Device/Method/Technique: Title page, abstract page, continuous text (Introduction, Description of Device/Method/Technique, Methods of Evaluation, Results of Evaluation, Discussion), Product Sources page, Acknowledgments page, references, tables, figure legends.
Case Report or Case Series: Title page, abstract page, continuous text (Introduction, Case Summary, Discussion), Acknowledgments page, references, tables, figure legends. Also see "How To Write a Better Case Report," Respir Care 1982;27:29 (Jan 1982).
Review Article: Title page, Table of Contents page, continuous text (Introduction, History, Review of Literature, State of the Art, Discussion, Summary), references. May include figures & tables. No abstract. Table of Contents optional. Other formats may be appropriate.
Overview, Update, Point of View, or Special Article: Title page, text (introduction, message), references, tables, figure legends. No abstract.
Letter: Title page (provide a title), text, writer's name & affiliation, references. Tables & figures may be included. Double-space everything. Write "For Publication" on title page.

Structure: Important Details

Title Page: List title of paper, all authors' full names, degrees, credential letters, professional positions, and affiliations. List correspondence address, telephone number, and reprint address if desired. Name sources of grants or other support. Identify any author's consulting or commercial relationships that pertain to the paper's topic.

Abstract Page: Number this Page 1. List paper's title but omit authors' names. Abstract should be 200 words or less and must be informative, briefly specifying main points of paper, such as methods, results, and conclusions drawn.

Statistical Analysis: In research articles, identify statistical tests and chosen level of significance in the Methods section. In Results section, report actual P values.

Figures (illustrations): All photographs, diagrams, & graphs must be numbered as Figure 1, Figure 2, etc, according to the order in which each is first mentioned in the text. Photographs must be glossy prints 5 × 7 to 8 × 10 inches and should be black & white unless color is essential. Letters and numerals must be neat and large enough to remain legible if figure is reduced in size for publication. Final figures must be of professional quality, but 'rough' sketches may accompany the submitted manuscript, with final figures to be prepared after review. Identify each figure on back with a stick-on label showing figure number and arrow indicating top; omit author's name. Cover label with clear tape so ink will not smudge other prints. Supply three sets of unmounted figures. If figure has been published before, include copyright-holder's written permission to use it.

Figure Legends: List figure legends on a separate page, not on figures. If a figure has been published before, list the source in the legend.

Tables: Type each table on a separate page. Avoid more than 8 columns across. Continue a deep table on following pages. Give each table a number and descriptive title, placed above the table. Double-space ALL lines in tables, including column headings and footnotes.

Drugs: Brand names may be given, but always also show generic names.

Units of Measurement: In addition to conventional units of measure, show SI values and units in brackets after conventional expressions: ie, "PEEP, 10 cm H_2O [0.981 kPa]." For conversion to SI, see RESPIRATORY CARE 1988;33:861-873 (Oct 1988).

Commercial Products: If three or fewer commercial products are named in the text, list the manufacturer's name and location in parentheses the first time each is mentioned. If four or more products are named, do not list manufacturers in the text; instead, name the products and manufacturers in a Products Sources list at the end of the text. Provide model numbers when available.

Abbreviations: Use an abbreviation only if the term occurs several times in the paper. Write out the full term the first time it appears, followed by the abbreviation in parentheses. Thereafter, employ the abbreviation alone. Never use an abbreviation without defining it. Do not create new abbreviations unless absolutely necessary.

References:

- Use references to support statements of fact, indicate sources of information, or guide readers to further pertinent literature.
- Cite only published works—or works accepted for publication. When listing an accepted but still unpublished work, designate the accepting journal's name, followed by "(in press)."
- In the text, cite references by superscript numerals (half space above text), not in parentheses. The first reference cited in the text is number 1, the next is number 2, etc.
- In the reference list, place the cited works in numerical order.
- For the reference list, obtain author names, article and book titles, dates, volume and page numbers from the *original* cited articles and books, not from secondary sources such as other articles' reference lists, which often are inaccurate.
- Type references in medical-journal style. Examples appear at the end of these Instructions. Abbreviate journal names as in *Index Medicus*. A list of many journal-name abbreviations was published in Respir Care 1988;33:1050 (Nov 1988).
- DOUBLE-SPACE the lines of references.
- List ALL authors' names. Do not use "et al" to substitute for names.
- Identify abstracts, editorials, and letters as such. See examples.

Personal Communications, Unpublished Papers, and Unpublished Observations: List unpublished items in parentheses in the text, not in the reference list.

Examples of How To Type References

Notes: Although the examples here are printed with single-spaced lines, please double-space references in manuscripts. Also, note that words in article and book titles are not capitalized—except proper names.

Standard Journal Article:

1. Shepherd KE, Johnson DC. Bronchodilator testing: An analysis of paradoxical responses. Respir Care 1988;33:667-671.

Corporate Author Journal Article:

2. American Association for Respiratory Care. Criteria for establishing units for chronic ventilator-dependent patients in hospitals. Respir Care 1988;33:1044-1046.

Article in Journal Supplement:

(Journals differ in their methods of numbering and identifying supplements. Supply sufficient information to allow retrieval.)

3. Reynolds HY. Idiopathic interstitial pulmonary fibrosis. Chest 1986;89(3, suppl):139s-143s.

Abstract in Journal:

(Abstracts are not strong references; when possible, full papers should be cited. When cited, abstracts should be identified as such.)

4. Lippard DL, Myers TF, Kahn SE. Accuracy of pulse oximetry in severely hypoxic infants (abstract). Respir Care 1988;33:886.

Editorial in Journal:

5. Rochester DF. Does respiratory muscle rest relieve fatigue or incipent fatigue? (editorial). Am Rev Respir Dis 1988;138:516-517.

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6. Smith DE, Herd D, Gazzard BG. Reversible bronchoconstriction with nebulised pentamidine (letter). Lancet 1988;2:905.

Personal Author Book:

7. Nunn JF. Applied respiratory physiology. New York: Appleton-Century-Crofts, 1969.

Note: To specify pages cited in a book, place a colon after the year and then list the page(s). Examples: 1969:85 (one page), 1963:85-95 (series of contiguous pages), 1963:85,95 (separated pages).

Corporate Author Book:

8. American Medical Association Department of Drugs. AMA drug evaluations, 3rd ed. Littleton CO: Publishing Sciences Group, 1977.

Book with Editor, Compiler, or Chairman as 'Author':

9. Guenter CA, Welch MH, eds. Pulmonary medicine. Philadelphia: JB Lippincott, 1977.

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10. Pierce AK. Acute respiratory failure. In: Guenter CA, Welch MH, eds. Pulmonary medicine. Philadelphia: JB Lippincott, 1977:171-223.

Submitting the Manuscript

After preparing the manuscript according to these Instructions, perform a final proofreading and check for accuracy and completeness. Then mail three copies of the manuscript and three sets of figures to RESPIRATORY CARE, 11030 Ables Lane, Dallas TX 75229 (or Federal Express to RESPIRATORY CARE, 11030 Ables Lane, Dallas TX 75229). Manuscript copy on IBM-compatible or Macintosh disks in addition to the requisite three hard copies will facilitate processing (Macintosh preferred). Enclose a letter as specified under **General Requirements** at the beginning of these Instructions. Do not submit material that has been published or is being considered elsewhere.

Author's Checklist

1. Is paper for a listed publication category?
2. Does cover letter meet specifications?
3. Is title page complete?
4. Are all pages double-spaced and numbered?
5. Are all references, figures, and tables cited in the text?
6. Are references typed in requested style?
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9. Has manuscript been proofread by all authors?

Notices

Notices of competitions, scholarships, fellowships, examination dates, new education programs, and the like will be listed here free of charge. Items for the Notices section must reach the Journal 60 days before the desired month of publication (January 1 for the March issue, February 1 for the April issue, etc). Include all pertinent information and mail notice to RESPIRATORY CARE Notices Dept, 11030 Ables Lane, Dallas TX 75229-4593.

THE ALLEN AND HANBURYS PUBLICATIONS AWARDS FOR 1991

1. $2,000 for the best original paper (study, evaluation, or case report) accepted for publication in RESPIRATORY CARE from November 1990 through October 1991.

2. Four awards of $1,000 each for papers based on 1990 OPEN FORUM presentations accepted for publication in RESPIRATORY CARE from November 1990 through October 1991.

3. Five awards of $500 each for the best papers (submitted, not necessarily published) from 'never published in RC' OPEN FORUM participants. The author must present the abstract at the Annual Meeting and must submit a paper based on the abstract *before* the Annual Meeting (received in the Editorial Office by December 1).

REGISTRATION REIMBURSEMENT FOR OPEN FORUM PAPERS

Any 1991 OPEN FORUM presenter (or co-author designee) who submits an adequately prepared paper based on his or her OPEN FORUM presentation *prior to* or *at* the 1991 Annual Meeting will be reimbursed for Annual Meeting registration. The submitted paper must contain complete data, be appropriately organized, and be typed double-spaced. Camera-ready artwork need not be submitted with the paper, but sketches of proposed figures should be clear and detailed enough to allow critique.

RADIOMETER AMERICA

Radiometer America Inc is offering three awards of $333 each for the best features from (1) Test Your Radiologic Skill, (2) Blood Gas Corner, and (3) PFT Corner accepted for publication from November 1990 through October 1991.

The awards will be made at the 1991 Annual Meeting. Papers are judged automatically. No application is necessary.

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1991—Atlanta, Georgia, December 7-10

1992—San Antonio, Texas, December 12-15

1993—Nashville, Tennessee, December 11-14

1994—Las Vegas, Nevada, December 12-15

1995—Orlando, Florida, December 2-5

PUBLICATION OF INTEREST

"A Summary of the Device User Facility Reporting Requirements of the Safety Medical Devices Act of 1990." (Public Law 101-629.) Rockville MD: Center for Devices and Radiological Health, 1990.

Single copies of this document are available from the United States Pharmacopeia (USP), 12601 Twinbrook Parkway, Rockville MD 20852.

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Applications Accepted Beginning:	July 1, 1991
Application Deadline:	September 1, 1991

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101	102	103	104	105	106	107	108	109	110	111	112	113	114	115	116	117	118	119	120
121	122	123	124	125	126	127	128	129	130	131	132	133	134	135	136	137	138	139	140
141	142	143	144	145	146	147	148	149	150	151	152	153	154	155	156	157	158	159	160

I. Type of Instn/Practice
1 ☐ Hosp. 500 or more beds
2 ☐ Hosp. 300 to 500 beds
3 ☐ Hosp. 200 to 300 beds
4 ☐ Hosp. 100 to 200 beds
5 ☐ Hosp. 100 or less beds
6 ☐ Clinic/Group Practice
7 ☐ Independent RT Provider
8 ☐ Industry (Mfgr/Sales)

II. Department
A ☐ Respiratory Ther
B ☐ Cardiopulmonary
C ☐ Anesthesia Service
D ☐ Emergency Dept.

III. Specialty
1 ☐ Clinical Practice
2 ☐ Perinatal Pediatrics
3 ☐ Critical Care
4 ☐ Clinical Research
5 ☐ Pulmonary Func Lab
6 ☐ Home Care/Rehab
7 ☐ Education
8 ☐ Management

IV. Position
A ☐ Dept. Head
B ☐ Chief Therapist
C ☐ Supervisor
D ☐ Staff Technician
E ☐ Staff Therapist
F ☐ Educator
G ☐ Medical Director
H ☐ Anesthesiologist
I ☐ Other MD
J ☐ Nurse

V. Are you a member of the AARC?
1 ☐ Yes
2 ☐ No

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61	62	63	64	65	66	67	68	69	70	71	72	73	74	75	76	77	78	79	80
81	82	83	84	85	86	87	88	89	90	91	92	93	94	95	96	97	98	99	100
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121	122	123	124	125	126	127	128	129	130	131	132	133	134	135	136	137	138	139	140
141	142	143	144	145	146	147	148	149	150	151	152	153	154	155	156	157	158	159	160

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H ☐ Anesthesiologist
I ☐ Other MD
J ☐ Nurse

V. Are you a member of the AARC?
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2 ☐ No

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